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MINING & WATER QUALITY:

TOXICITY OF POTENTIAL WATER CONTAMINANTS

Prepared for:

United States Department of Agriculture
Forest Service
Rocky Mountain Forest & Range Experiment Station

Prepared by:

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INTRODUCTION

Purpose

The U.S. Forest Service is investigating the impact on water quality of past and present mining activities on Forest Service lands. During Phase I of the investigation, a list of potential contaminants from mining and processing was developed. Wright Consultants, Inc. (WCI) was tasked to conduct Phase II of the investigation, which entailed a comprehensive literature review to assess the toxicity of the potential water contaminants identified in Phase I.

In Phase I of this assessment of potential effects of mining on water quality, mining location data were acquired from the U.S. Bureau of Mines, along with administrative and watershed boundaries. These data are currently being evaluated for their inclusion in a publication describing the distribution of abandoned and inactive mines nationwide. It is recognized that a variety of chemical compounds may end up in surface water as a result of metallic, nonmetallic and energy extraction activities. A list of potential contaminants from mining and processing has been developed under contract and will be published separately.

Phase II of this project encompassed several areas. The purpose was to determine, via comprehensive literature review, the toxicity of the potential water contaminants from mining (native elements and compounds) that were identified in the above-mentioned study. A parallel effort is being undertaken to add coal data to the MASNP database. This report presents the results of the Phase II study.

Methods of Investigation

WCI identified a number of major reference sources (including both printed and on-line computer databases) providing toxicological effects data primarily regarding human exposure. These include the Agency for Toxic

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Substances and Disease Registry (ATSDR) Toxicologic Profiles (1a-p), Health Effects Assessment Summary Tables (HEAST) (2), Hazardous Substances Data Base (HSDB) (3), EPA's Integrated Risk Information System (IRIS) (4), Material Safety Data Sheets (MSDS) (5), Registry of Toxic Effects Chemical Substances (RTECS) (6), Sax (7), McNulty (8), and EPA's "Gold Book". IRIS, HSDB and RTECS are online computer databases which were accessed by WCI. Full references for these sources are provided in the Bibliography section of this report. Sources are referenced by number throughout the remainder of the body of the text of this report.

Types of Data

A significant amount of the toxicological database is anecdotal or case study information (e.g., exposure to concentrations of 250 ppm of sodium hydrosulfide has been shown to cause irritation of mucous membranes, bronchitis and pulmonary edema). Because this information is not standardized, it does not lend itself to making comparisons of the toxicity of different chemicals, however, it is useful as a general measure of the types of health effects that may be expected at certain exposure levels and through various exposure routes (e.g, inhalation, ingestion, dermal, subcutaneous).

Much of the toxicological data also derive from laboratory animal studies conducted to determine lethal doses of chemicals, as well as health effects at below-lethal doses. Common reporting parameters for animal studies are LD50s (a calculated dose of a substance which is expected to cause the death of 50% of an experimental animal population), and LC50s (a calculated concentration of a substance in air which is expected to cause the death of 50% of an animal population after exposure for a specified length of time). Different types of experimental animals, as well as different exposure routes are often used in these studies, thus while the data are useful as a measure of toxicity, they do not allow easy cross-comparisons between chemicals.

There are numerous other measures of toxicity reported in the literature (e.g., RfCs, RfDs, NOAELs, LOAELs). These and a host of other toxicological acronyms are defined in the Glossary section of this report. In addition to toxicity measurements, various regulatory standards and environmental indices have been established for some of the chemicals of interest. These reference standards do provide some basis of comparison for the chemicals. Those of most interest include EPA's carcinogenicity groups, drinking water standards, and ambient water quality criteria. These standards are toxicologically based.

For example, Section 305(a)(1) of the Clean Water Act requires EPA to publish and periodically update ambient water quality criteria. These criteria are to accurately reflect the latest scientific knowledge (a) on the kind and extent of all identifiable effects on health and welfare including, but not limited to, plankton, fish, shellfish, wildlife, plant life, shorelines, beaches, aesthetics, and recreation which may be expected from the presence of pollutants in any body of water including groundwater; (b) on the concentration and dispersal of pollutants, or their by-products, through biological, physical, and chemical processes; and (c) on the effects of pollutants on biological community diversity, productivity, and stability, including information on the factors affecting rates of eutrophication and organic and inorganic sedimentation for varying types of receiving waters. These criteria are not rules and they do not have regulatory impact. Rather, these criteria present scientific data and guidance on the environmental effects of pollutants which can be used to derive regulatory requirements based on considerations of water quality impacts (9).

Data Limitations

Substantial volumes of toxicological information exist for certain chemicals. For example, chemicals that are in common use or are known to be highly toxic have been extensively studied. The databases for many of the metals, and organics such as creosote are very large. Data for the more esoteric chemicals such as some of the xanthates are not readily available. Thus the volume of information presented in the appendices to this report varies significantly from chemical to chemical. In addition, as discussed above, most of the data available derive from human case studies or laboratory animal studies. There is very little information

available on toxicity to fish and wildlife, and ambient water quality criteria have been developed for only a limited number of chemicals.

Data Presentation

The format and content of the report was determined in meetings and discussions with U.S. Forest Service personnel. It was decided that a summary sheet of toxicological information would be presented for each chemical. The chemicals are presented in alphabetical order, within three groupings: inorganics, organics (divided into xanthates, dithiophosphates, and other organics), and non compound-specific organics. For each chemical, general information on chemical origin and usage is presented, followed by a brief overview of data availability. A synopsis of available toxicological information regarding effects on humans and other species is then provided. Excerpts from Sax are included if available. Reference standards including human carcinogenicity group, drinking water standards, and water quality criteria are identified if found in the literature. Following the chemical summaries is the Glossary. This is followed by the Bibliography. Appendices are provided separately. These include all the printouts from the online computer search as well as copies of certain informative printed materials. The reader is referenced to these appendices for further information on the chemicals, including toxicological measures such as LD50s and LC50s that are not presented in the text.

INORGANICS

Arsenic

Arsenic (syn. ARSENICALS; ARSENIC-75; ARSENIC BLACK; COLLOIDAL ARSENIC; METALLIC ARSENIC), has a CAS number of 7440-38-2. Arsenic is a naturally-occurring element. There are two forms of arsenic - organic and inorganic. Organic forms are usually less harmful than inorganic forms. Inorganic arsenic occurs naturally in many kinds of rock, especially ores that contain copper or lead. When these ores are heated at smelters to get the copper or lead, most of the arsenic enters the air as a fine dust. Smelters collect this dust and purify the arsenic for several uses. The main use is as a preservative for wood to make it resistant to rotting and decay. Arsenic is also used to make several types of insect killers. Most inorganic and organic arsenic compounds are white or colorless powders that do not evaporate. They have no smell and no special taste, therefore, they cannot be detected in food, water or air (1a).

Information pertaining to arsenic has been obtained from ATSDR (1a), HEAST (2), HSDB (3), IRIS (4), RTECS (6), and Sax (7). Arsenic has been well researched and its toxicity toward humans and other species comparatively well-established. Information available from the above-referenced databases includes various LD50s, TDLos, OSHA PELs, ACGIH TLVs, etc. (see Appendices).

Inorganic arsenic has been recognized as a human poison since ancient times. If ingested in large doses, irritation of the stomach occurs, with symptoms such as pain, nausea, vomiting and diarrhea. Other effects include decreased production of red and white blood cells and impaired nerve function. Inhalation of arsenic causes sore throat, lung irritation, skin effects and an increase in the risk of lung cancer. HHS has determined that arsenic and certain arsenic compounds are known carcinogens. People living near smelters, chemical factories, or waste sites with arsenic may have increased risk of lung cancer as well. Small amounts of arsenic in the normal diet (10-50 ppb) may be beneficial to health, however, no cases of arsenic deficiency in humans have been reported (1a). Some information on fish and seafood concentrations of arsenic was available from case studies profiles in HSDB, and an aquatic life summary was prepared by EPA (see Appendices).

Sax summarizes the data as: A human carcinogen. Poison by subcutaneous, intramuscular, and intraperitoneal routes. Human systemic skin and gastrointestinal effects by ingestion. An experimental teratogen and tumorigen. Mutagenic data. Flammable in the form of dust when exposed to heat or flame or by chemical reaction with powerful oxidizers (7).

Reference standards found in the literature included the following:
Human Carcinogenicity Group: A
Drinking Water Standard (MCL): 0.05 mg/L
Water Quality Criteria - freshwater: 4-day ave. conc. As III not to exceed 190 ug/L on ave. and 1-hour ave. conc. not to exceed 360 ug/L more than once every 3 years
- saltwater: 4-day ave. conc. As III not to exceed 36 ug/L on ave. and 1-hour ave. conc. not to exceed 69 ug/L more than once every 3 years.

Arsenic Pentasulfide

Arsenic pentasulfide has the CAS number of 1303-34-0. It is used as a depressant in the flotation of molybdenum sulfides (8). No information was available concerning its manufacture. It exists as a brownish-yellow glassy, amorphous, highly refractive mass (7).

Information pertaining to arsenic pentasulfide has been obtained from Sax only (7). Arsenic compounds in general are highly toxic (see Arsenic in the appendices). No information concerning toxicity toward aquatic species, birds, or plants could be found.

Sax summarizes the data as: Flammable in the form of dust when exposed to heat or flame. Explosive when intimately mixed with powerful oxidizers, such as chlorine gas. Will react with water and steam to produce toxic and flammable vapors. Incompatible with water, steam, and strong oxidizers (7).

No reference standards were found in the literature.

Bismuth

Bismuth (syn. BISMUTH-209), has a CAS number of 7440-69-9. Bismuth is a naturally occurring element. It is obtained as a by-product from refining domestic lead and imported bullion. It is also obtained from the copper smelting and refining process and as a by-product of tin ore processing. It is used in the manufacture of bismuth salts, stereotype metal, fusible boiler plugs, electric fuses and low-melting solders; in tempering baths for steel, "silvering" mirrors and in dental techniques. It's primary uses, however, are in pharmaceuticals and cosmetics (3).

Information pertaining to bismuth has been obtained from HSDB (3), RTECS (6), and Sax (7). Bismuth has not been extensively researched and the data base on its toxicity toward humans and other species is modest. Information available from the above-referenced sources includes various LD50s and an LDLo (see Appendices).

Among the health effects reported are nausea, vomiting and diarrhea in both chronic and acute cases, myocardial damage, ulcerative stomatitis, gum discoloration, confusion, nephritis and renal failure, allergic contact dermatitis, and a number of psychiatric symptoms following bismuth intoxication (3). No data were found on toxicity to plants, aquatic animals or birds.

Sax data summaries on bismuth compounds includes the following: Bismuth and its salts can cause kidney damage, although the degree of such damage is usually mild. Large doses can be fatal. Industrially it is considered one of the less toxic of the heavy metals, although intoxication has occurred from its use in medicine. Death of animals from bismuth nephritis following injections of soluble salts occurs within several hours to 24 days. All bismuth compounds do not have equal toxicity (7).

No reference standards were found in the literature.

Cadmium

Cadmium (syn. COLLOIDAL CADMIUM; KADMIUM (GERMAN), has a CAS number of 7440-43-9. Cadmium is naturally occurring in many sources, including greenockite ore and lead and copper ores containing zinc. Coal and other fossil fuels contain cadmium and their combustion releases the element into the environment. Volcanic action is considered to be the major natural source of cadmium. It is liberated during smelting and refining and is used in various alloys. Among it's other uses are coating telephone cables, welding, sprinkling systems, fire alarms, starting switches, paint and pigment manufacture, coloring of plastics and ceramic glazes, electroplating, and in chemical synthesis (3).

Health effects information on cadmium can be found in ATSDR (1b), HEAST (2), HSDB (3), IRIS (4), RTECS (6), and Sax (7). Cadmium has been well researched and its toxicity toward humans and other species comparatively well-established. Information available from the above-referenced databases includes various LD50s, TDLos, OSHA PELs, ACGIH TLVs, etc. (see Appendices).

Cadmium has no known good effects on health. Breathing air with very high levels of cadmium can cause severe lung damage and death. Inhalation of lower levels of cadmium over a period of years can cause kidney disease. HHS has determined that cadmium and certain cadmium compounds may reasonably be anticipated to be carcinogens. Ingestion of food or water with very high cadmium levels severely irritates the stomach, leading to vomiting and diarrhea. Ingesting lower levels over a long period of time can cause kidney damage. There are no known health effects resulting from skin contact with cadmium. HSDB provides no data on plant, fish, seafood or animal concentrations, however, EPA has established water quality criteria as provided below.

Sax summary data includes: A human poison by inhalation and possibly other routes. Poison experimentally by ingestion, inhalation, intraperitoneal, subcutaneous, intramuscular, and intravenous routes. An experimental carcinogen, tumorigen, neoplastigen, and teratogen. Experimental reproductive effects. Mutagenic data. The dust ignites spontaneously in air and is flammable and explosive when exposed to heat, flame, or by chemical reaction with oxidizing agents and other substances. Explodes on contact with hydrazoic acid. Violent or explosive reaction when heated with ammonium nitrate. Vigorous reaction when heated with nitryl fluoride. When heated strongly it emits toxic fumes of cadmium (7).

Reference standards found in the literature included the following:
Human Carcinogenicity Group: B1
Drinking Water Standard (MCL): 0.005 mg/L
Water Quality Criteria - freshwater: 4-day ave. conc. not to exceed e(0.7852[1n(hardness)]-3.490)ug/L on ave. and 1-hour ave. conc. not to exceed e(1.128[1n(hardness)]-3.828ug/L more than once every 3 years
- saltwater: 4-day ave. conc. not to exceed 9.3 ug/L on ave. and 1-hour ave. conc. not to exceed 43 ug/L more than once every 3 years.

Calcium Carbide

Calcium carbide (syn. ACETYLENOGEN; CALCIUM ACETYLIDE; CALCIUM DICARBIDE; etc.), has the CAS number of 75-20-7. It has a variety of uses, including as a reducing agent, in fire signals; in welding and cutting materials; as a chemical intermediate for acetylene; in the production of the fertilizer calcium cyanamide; as a pineapple flowering agent; and in the generation of light in mine lamps (3,8). It is made primarily from the interaction of pulverized limestone or quicklime with crushed coke or anthracite in an electric furnace. It usually consists of grayish-black lumps with a garlic-like odor (3). It decomposes in both cold and hot water, yielding acetylene gas.

Information pertaining to calcium carbide has been obtained from the HSDB (3), RTECS (6), and Sax (7). Information available (see appendices) available from the above-referenced databases does not include LD50 and TDLo studies. The principle danger in human exposure to calcium carbide is the strong likelihood of severe burns upon contact with skin, eyes, and mucous membranes. No information concerning toxicity toward aquatic species, birds, or plants could be found.

Sax summarizes the data as: Reaction on contact with moisture from explosive acetylene gas. Flammable on contact with moisture, acid or acid fumes; evolves heat or flammable vapors. Moderate explosion hazard...(7).

No reference standards were found in the literature.

Calcium Chloride

Calcium chloride (syn. CALPLUS, CALTAC, DOWFLAKE, LIQUIDOW, PELADOW, SNOMELT, SUPERFLAKE ANHYDROUS), has a CAS number of 10043-52-4. Calcium chloride is used for antifreeze and refrigerating solutions, in fire extinguishers, as a wood preservative, in glues and cements, fireproofing fabrics, as a coagulant in rubber manufacture, etc. It is also used as a dust controller in road construction and mining operations, and as a component of oil and gas well fluids (e.g., drilling muds). Other uses are as an additive in herbicides to control the growth of vegetation (3).

Information pertaining to calcium chloride has been obtained from HSDB (3) and Sax (7). Calcium chloride has not been extensively researched and data are limited. Available information includes various LD50s and TDLos (see Appendices).

Calcium chloride causes coughing and wheezing if inhaled. Ingestion effects include nausea, vomiting and diarrhea. Dermatologic effects include redness, swelling and pain. Calcium chloride was found to be a powerful irritant on skin and mucous membranes. Acute ingestion of calcium chloride may produce significant gastrointestinal irritation and/or hemorrhage. In countries where calcium chloride is used instead of salt to melt snow on roads, there have been serious losses among wild animals drinking slush at roadside. Fish kills due to exposure to various concentrations of calcium chloride have been reported. These have included rock bass, bluegill, sunfish, mosquito fish, pickerel fry, whitefish fry and others. In addition, high concentrations of calcium chloride in irrigation water will reduce plant growth (3).

Sax summarizes the data as: Poison by intravenous, intramuscular, intraperitoneal, and subcutaneous routes. Moderately toxic by ingestion. An experimental tumorigen. Mutagenic data. Reaction with zinc releases explosive hydrogen gas. Exothermic reaction with water. When heated to decomposition, it emits toxic fumes of chlorine (7).

No reference standards were found in the literature.

Calcium Hydroxide

Calcium hydroxide (syn. BELL MINE; CALCIUM HYDRATE; HYDRATED LIME; LIME WATER; SLAKED LIME; etc.), has the CAS number of 1305-62-0. It has a variety of uses, including in the manufacture of lime paints; purification of sugar juices; as a food additive; as a disinfectant; in the treatment of fluoride poisoning; as an antacid for farm animals; and as a depressant in mineral ore flotation (3,8). It is prepared commercially by the hydration of lime. It usually consists of colorless to white odorless crystals or powder with a slightly bitter alkaline taste (3). It is soluble in water and inorganic acids, and insoluble in alcohol and most organic solvents.

Information pertaining to calcium hydroxide has been obtained from the HSDB (3), RTECS (6), and Sax (7). Information available (see appendices) available from the above-referenced databases includes several LD50 studies. The principle danger in human exposure to calcium hydroxide is the strong likelihood of severe burns upon contact with skin, eyes, and mucous membranes. However, ingestion of large amounts may lead to acute poisoning, with severe pain, vomiting, diarrhea leading to death in severe cases. The FDA affirms that calcium hydroxide is generally recognized as a safe direct human food ingredient (3). Little information concerning toxicity toward aquatic species, birds, or plants could be found. The HSDB reports that calcium hydroxide does not degrade oxidatively; it is neutralized by absorption of atmospheric carbon dioxide. Calcium hydroxide shows no bioaccumulation or food chain concentration toxicity potential (3).

Sax summarizes the data as: Mildly toxic by ingestion. A severe eye irritant. A skin, mucous membrane and respiratory system irritant. Mutagenic data. Causes dermatitis. Dust is considered to be a significant industrial hazard. A common air contaminant...(7).

No reference standards were found in the literature.

Cobalt

Cobalt (syn. NONE), has a CAS number of 7440-48-4. Cobalt is a naturally occurring element from several mineral sources. It occurs as a by-product or coproduct of the refining of other mined metals such as copper and nickel. The amount of cobalt mined is relatively small in comparison with copper and nickel, and the cobalt supply depends to a large extent on the demand for the latter two metals. Cobalt molybdenum alloys are used for the desulfurization of high-sulfur bituminous coal and cobalt iron alloys in the hydrocracking of crude oil shale and in coal liquefaction. Cobalt is also used in nuclear technology; in the manufacture of magnets; in tools, cars, electrical and aircraft industries; in the production of cobalt-bearing alloys, cutting materials, wear-resistant materials, lacquers, varnishes, paint driers; production of inks, enamels, frits, glazes; and in gamma ray therapy of carcinoma.

Information pertaining to cobalt has been obtained from ATSDR (1c), HSDB (3), IRIS (4), RTECS (6), and Sax (7). The chemical has been comparatively well researched and its toxicity toward humans and other species has been generally established. Information available from the above-referenced databases includes various LDLos, TDLos, OSHA PEL, ACGIH TLV, etc. (see Appendices).

Occupational inhalation of cobalt can produce interstitial fibrosis ("hard metal disease") Ingestion or inhalation of cobalt causes nausea, vomiting, diarrhea, and colicky abdominal pain. Other health effects include pulmonary changes in exposed workers, deterioration of the sense of smell and decreased blood pressure. There is also evidence of bronchitis, emphysema or asthma in workers exposed to cobalt in the hard metal industry. NIOSH considers the information available sufficient to conclude that cobalt metal and salts, and probably cobalt oxides and a number of organo-cobalt complexes, can damage the kidneys (3).

Only a few plants accumulate cobalt above concentrations that cause severe phytotoxicity (3). HSDB reports no data available for plant, fish, seafood or animal concentrations.

Sax summary data includes the following: Poison by intravenous, intratracheal, and intraperitoneal routes. Moderately toxic by ingestion. An experimental neoplastigen and tumorigen. Inhalation of the dust may cause pulmonary damage. The powder may cause dermatitis. Ingestion of soluble salts produces nausea and vomiting by local irritation. Powdered cobalt ignites spontaneously in air. Flammable when exposed to heat or flame (7).

Reference standards found in the literature included the following:
Human Carcinogenicity Group: B2

Copper

Copper (syn. ALLBRI NATURAL COPPER; ANAC 110; ARWOOD COPPER; BRONZE POWDER; CDA 101; CDA 102; CDA 110; CDA 122; C.I. 77400; C.I. PIGMENT METAL 2; COPPER-AIRBORNE; COPPER BRONZE; COPPER-MILLED; COPPER SLAG-AIRBORNE; COPPER SLAG-MILLED; 1721 GOLD; GOLD BRONZE; KAFAR COPPER; M1 (COPPER); M2 (COPPER); OFHC Cu; RANEY COPPER), has a CAS number of 7440-50-8. Copper is a naturally occurring element present in many minerals. The main source of elevated copper levels in the air is copper dust from copper processing operations. Smelting operations may produce elemental copper and it is likely that municipal incineration will also produce copper. Copper is used in heating, chemical and pharmaceutical machinery; alloys; electroplated protective coating; corrosion-resistant piping; catalyst; insulation for liquid fuels; and in the construction, industrial machinery and equipment, and transportation industries (3).

Information pertaining to copper has been obtained from ATSDR (1d), HEAST (2), HSDB (3), IRIS (4), RTECS (6), Sax (7), and EPA (9). Copper has been well researched and its toxicity toward humans and other species comparatively well-established. Information available from the above-referenced databases includes various LD50s, TDLos, ACGIH TLVs, etc. (see Appendices).

Ingestion of copper causes gastrointestinal irritation and associated emesis. Inhalation of copper dust or fumes may cause a metal fever syndrome or respiratory signs such as wheezing. Skin exposure may result in severe irritation. The literature indicates that it is debatable whether chronic copper poisoning, industrial or nonindustrial, exists in human beings. Many cases of illness formerly attributed to copper are now believed to have been more probably due to admixture with other metals, especially lead. The fate of copper with respect to its leachability in purely organic spruce forest soils was studied. Appreciable mobilization of copper occurred only with prolonged leaching at Ph 2.8, therefore it does not appear likely that acidic rainfall will result in significant mobilization of copper from organic soils unless the rainfall Ph drops below 3 (3). HSDB reports that no data are available on fish/seafood concentrations, however an aquatic life summary was prepared by EPA (see Appendices).

Sax provides summary data including the following: An experimental tumorigen and teratogen. Other experimental reproductive effects. Human systemic effects by ingestion; nausea and vomiting. Liquid copper explodes on contact with water (7).

Reference standards found in the literature included the following:
Human Carcinogenicity Group: D
Drinking Water Standard (MCL): 1.3 mg/L
Water Quality Criteria - freshwater: 4-day ave. conc. not to exceed e(0.9422[1n(hardness)]-1.464)ug/L and 1-hour ave. conc. not to exceed e(0.9422[1n(hardness)-1.464)ug/L more than once every 3 years
- saltwater: 1-hour ave. conc. not to exceed 2.9 ug/L more than once every 3 years

Copper Sulfate

Copper sulfate (syn. BCS COPPER FUNGICIDE; BLUE COPPER; BLUE STONE; BLUE VITRIOL; COPPER MONOSULFATE; SP BASIC SULFATE; CUPRIC SULFATE; KUPPERSULFAT (GERMAN); ROMAN VITRIOL; SULFATE DE CUIVRE (FRENCH); SULFURIC ACID, COPPER (2+) SALT (1:1); TNCS 53; TRIANGLE) has a CAS number of 7758-98-7. Copper sulfate occurs in nature as the mineral hydrocyanite. Other sources include mining and ore crushing and beneficiation processes, copper processing, smelting operations, tobacco smoke, and stack emissions of coal-burning power plants. Among its uses are asphalt-making, battery-making, electroplating, fungicide production, gem coloring, lithography, soldering, water treatment, and wood preservation (3).

Information pertaining to copper sulfate has been obtained from HSDB (3), RTECS (6), and Sax (7). Copper sulfate has been moderately researched and its toxicity to humans and other animals generally established. Information available from the above-referenced databases includes various LD50s, LDLos, TDLos, OSHA PEL, ACGIH TLV, etc. (see Appendices).

Cases of acute poisoning (some of them fatal) from ingesting copper sulfate have been documented. Symptoms included repeated vomiting, diarrhea, blood in the gastrointestinal tract and ulceration. Skin contact can result in itching exzema and eye contact can result in conjunctivitis, edema of the eyelids and ulceration and turbidity of the cornea.
Inhalation of dust and mists of copper salts can result in irritation of nasal mucous membranes. Copper, an essential nutrient, is strongly bioaccumulated but not biomagnified. With regard to plant concentrations, it is considered more probable that contamination persists longer than is generally thought possible. No data are available concerning fish/seafood concentrations (3).

Sax provides summary data including the following: A human poison by ingestion. An experimental poison by ingestion, subcutaneous, parenteral, intravenous and intraperitoneal routes. An experimental tumorigen. Human systemic effects by ingestion: gastritis, diarrhea, nausea or vomiting, damage to kidney tubules, and hemolysis. Mutagenic data. When heated to decomposition it emits toxic fumes.

No reference standards were found in the literature.

Gold

Gold (syn. BURNISH GOLD; COLLOIDAL GOLD; GOLD FLAKE; GOLD LEAF; GOLD POWDER; MAGNESIUM GOLD PURPLE; SHELL GOLD), has a CAS number of 7440-57-5. Gold is naturally occurring in the free state as veins in quartz, in river sands and other sources. It is used in jewelry, dentistry and medicine. Gold is also used in infrared reflectors; electrical contact alloys; brazing alloys; polarographic electrodes; colloidal dispersions used in coloring glass. Over 50% of the gold produced is used for jewelry and art products (3).

Information concerning gold has been obtained from HSDB (3), RTECS (6), and Sax (7). Gold has not been extensively researched and information on its toxicity to humans and other species is limited. Information from the above-referenced databases includes LDLos, and TDLos (see Appendices).

Small amounts of gold have been reported in many foods. Among the health effects reported as a result of excessive exposure to gold are stomatitis, gingivitis, ocular inflammation, ocular ulcers, stroke, diarrhea and severe colitis. Dermatologic reactions to gold salts are the most common effects (3). Animal studies show tumorigenic effects and acute pulmonary edema (6). Gold is not normally present in plant tissues and it is not an essential nutrient. No data were found for fish/seafood or animal concentrations (3).

Sax summary data includes the following: Poison by intravenous route. An experimental tumorigen. Experimental reproductive effects. Can form explosive compounds.

No reference standards were found in the literature.

Hydrogen Fluoride (Hydrofluoric Acid)

Hydrogen fluoride (syn. ACIDE FLUORHYDRIQUE (FRENCH); ACIDO FLUORIDRICO (ITALIAN); ANHYDROUS HYDROFLUORIC ACID (DOT); FLUOROWODOR (POLISH); FLUORWASSERSTOFF (GERMAN); FLUORWATERSTOF (DUTCH); HYDROFLUORIC ACID, ANHYDROUS (DOT); HYDROFLUORIC ACID SOLUTION (DOT); HYDROFLUORIDE; HYDROGEN FLUORIDE (ACGIH, DOT); RCRA WASTE NUMBER U134), has a CAS number of 7664-39-3. Sources of hydrogen fluoride include cooling and gypsum settling ponds associated with the manufacture of phosphate fertilizer. Hydrogen fluoride is used for cleaning cast iron, copper and brass; removing efflorescence from brick and stone or sand particles from metallic castings; frosting, etching glass and enamel; polishing crystal glass; and enameling and galvanizing iron.

Information pertaining to hydrogen fluoride has been obtained from HSDB (3), IRIS (4), RTECS (6), and Sax (7). Hydrogen fluoride has not been extensively researched and the data base on its toxicity to humans and other species is limited. IRIS indicates that risk assessments including an oral RfD assessment and an inhalation RfC assessment are currently under review by EPA work groups. Information available from the above-referenced databases includes various LC50s, LDLos and TCLos, as well as an OSHA PEL and ACGIH TLV (see Appendices).

Hydrogen fluoride is highly corrosive and may cause systemic fluoride poisonings. Eye exposure results in severe ocular damage. Severe chemical pneumonitis may occur following inhalation of hydrogen fluoride fumes. Ingestion of hydrofluoric acid results in vomiting and painful lesions. It is highly corrosive to skin and mucous membranes. The sources reviewed provide no data on plant, fish/seafood or animal concentrations.

Sax provides summary data on hydrogen fluoride including the following: A human poison by inhalation. A poison experimentally by inhalation, subcutaneous and intraperitoneal routes. A corrosive irritant to skin, eyes (at 0.05 mg/L), and mucous membranes. Experimental teratogenic effects. Experimental reproductive effects. Mutagenic data. Inhalation of the vapor may cause ulcers of the upper respiratory tract. Concentrations of 50-250 ppm are dangerous, even for brief exposures. Hydrofluoric acid produces severe skin burns which are slow in healing. The subcutaneous tissues may be affected, becoming blanched and bloodless. Gangrene of the affected areas may follow. It is a common air contaminant. It has explosive and violent reactions with other chemicals. There is a dangerous storage hazard with nitric acid and other compounds. Reacts with water or steam to produce toxic and corrosive fumes. When heated to decomposition it emits highly corrosive fumes of fluorine.

No reference standards were found in the literature.

Iron

Iron (syn. ANCOR EN 80/150; ARMCO IRON; CARBONYL IRON), has a CAS number of 7439-89-6. Iron is the second most abundant metal in the earth's crust. Among its many uses are in the manufacture of iron and steel castings; as a material to increase the density of oil well drilling fluids; as an alloy with many other elements; furnace steelmaking and foundry operation; as a coolant in Bessemer-oxygen furnace steelmaking; as a replacement for scrap in the open hearth and in various smelting processes. Mining and handling of iron ores provides exposures to dusts of iron oxides (3). The ferrous form can persist in waters void of dissolved oxygen and originates usually from groundwaters or mines when these are pumped or drained (9).

Information pertaining to iron has been obtained from HEAST (2), HSDB (3), RTECS (6), Sax (7) and EPA (9). Iron has been well researched and its toxicity toward humans and other species comparatively well-established. Information available from the above-referenced databases includes various LD50s, TDLos, and LDLos (see Appendices).

Iron is reportedly harmful only under unusual conditions or overwhelming dosage. Long-term inhalation exposure to iron has resulted in mottling of the lungs, a condition referred to as siderosis. This does not ordinarily cause significant physiologic impairment (3).

Iron is an essential trace element required by both plants and animals. In some waters, it may be a limiting factor for the growth of algae and other plants. It is a vital oxygen transport mechanism in the blood of all vertebrate and some invertebrate animals.

Sax provides summary data including the following: Poison by intraperitoneal route. Iron is potentially toxic in all forms and by all routes of exposure. The inhalation of large amounts of iron dust results in "arc welders lung". Chronic exposure to excess levels of iron (greater than 50-100 mg iron/day) can result in pathological deposition of iron in the body tissues, the symptoms of which are fibrosis of the pancreas, diabetes mellitus, and liver cirrhosis. As with other metals, it becomes more reactive as it is more finely divided. Ultrafine iron powder is potentially explosive.

Reference standards found in the literature included the following:
Water Quality Criteria: 0.3 mg/L for domestic water supplies (welfare)
1.0 mg/L for freshwater aquatic life

Lead

Lead (syn. C.I. 77575; C.I. PIGMENT METAL 4; GLOVER; LEAD FLAKE; LEAD S2; OLOW (POLISH); OMAHA; OMAHA & GRANT; SI; SO), has a CAS number of 7439-92-1. Lead is the fifth most important metal commercially in the U.S. and it may enter the environment during its mining, ore processing, smelting, refining use, recycling or disposal. Generally the initial means of entry is via the atmosphere. Over 70% of the lead metal consumed was used for batteries with the remainder used for gasoline antiknock additives, pigments and ceramics, ammunition, solder, cable covering, caulking, pip and sheet, type metal, brass and bronze, bearings and miscellaneous (3).

Information pertaining to lead has been obtained from HEAST (2), HSDB (3), IRIS (4), RTECS (6), Sax (7), and EPA (9). Lead has the most extensive database of any chemical researched and its toxicity toward humans and other species is well-established. Information available from the above-referenced databases includes various LDLos, TCLos, TDLos, OSHA PEL, ACGIH TLV, etc. (see Appendices).

Data are available on acute and chronic toxicity of lead to fish and invertebrates, both freshwater and saltwater (9). Extensive studies of lead concentrations in fish/seafood have also been performed. Concentrations in ducks, waterfowl, other birds, snakes, frogs, muskrats and other species have also been reported (3).

Information included in the Sax summary of health effects of lead includes: Poison by ingestion. Moderately toxic by intraperitoneal route. It is a suspected carcinogen of the lungs and kidneys. Human systemic effects by ingestion and inhalation: loss of appetite, anemia, malaise, insomnia, headache, irritability, muscle and joint pains, tremors, flaccid paralysis without anesthesia, hallucinations and distorted perceptions, muscle weakness, gastritis and liver changes. The major organ systems affected are the nervous system, blood system, and kidneys. Lead in the blood has been found to diminish the IQ scores of children. Reversible kidney damage can occur from acute exposure. Severe toxicity can cause sterility, abortion and neonatal mortality and morbidity. An experimental teratogen. Experimental reproductive effects. Human mutagenic data. Lead effects the red blood cells and produces a damaging effect on the organs or tissues with which it comes in contact. Autopsies in deaths attributed to lead poisoning, and experimental work on animals have shown pathological lesions of various organs and tissues. Flammable and moderately explosive in the form of dust when exposed to heat or flame.

Reference standards found in the literature included the following:
Human Carcinogenicity Group: B2
Drinking Water Standard (MCLG): 0 mg/L
NAAQS: 1.5 ug/cu.m
Water Quality Criteria - freshwater: 4-day ave. conc. not to exceed e(1.273[1n(hardness)]-4.705) ug/L on ave. and 1-hour ave. conc. not to exceed e(1.273[1n(hardness)]-1.460 ug/L more than once every 3 years on ave.
- saltwater: 4-day ave. conc. not to exceed 5.6 ug/L and 1-hour ave conc. not to exceed 140 ug/L more than once every 3 years on ave.

Lead Oxides

Lead oxides (syn. LEAD MONOXIDE; LEAD TETROXIDE; LEAD OXIDE RED; C.I. 77578; C.I. PIGMENT RED 105; DILEAD [2] LEAD [IV] OXIDE; GOLD SATINOBRE; LEAD ORTHOPLUMBATE; LEAD TETRAOXIDE; MINERAL ORANGE; MINERAL RED; MINIUM; MINIUM NON-SETTING RL-95; ORANGE LEAD; PARIS RED; PLUMBOPLUMBIC OXIDE; RED LEAD; READ LEAD OXIDE; SANDIX; SATURN RED; TRILEAD TETROXIDE), have the CAS numbers 1314-41-6 and 1317-36-8. Lead oxide occurs naturally in lanarkite and other lead minerals in combination with other lead salts. Lead oxide is the dominant commercial compound of lead. Lead compounds enter the environment during mining ore processing, smelting, refining, use, recycling or disposal and the atmosphere is the major initial recipient. Lead oxides are used in pottery and enameling; purification of alcohol; as a pigment in anticorrosion paints for steel surfaces; in adhesives; in catalysts for combustion of carbon monoxide in exhausts; as radiation shields; etc. (3).

Information pertaining to lead oxides has been obtained from HSDB (3), RTECS (6), and Sax (7). Lead oxides have been moderately well researched and their toxicity toward humans and other receptors comparatively well-established. Information available from the above-referenced sources includes various LD50s, LDLos, OSHA PEL and ACGIH TLV (see Appendices).

The reader is referred to lead for many of the health and environmental effects discussed in the literature on lead oxides. Lead oxide will not leach in soil because of its relative insolubility. If released into water, lead oxide will partly settle out due to its fairly low solubility and partially dissolve.

Summary information provided by Sax for lead oxide red includes the following: Poison by intraperitoneal route. Moderately toxic by ingestion. Combustible by chemical reaction with reducing agents. An oxidizing agent. Explodes on contact with peroxyformic acid. When heated to decomposition, it emits toxic fumes of lead.

No reference standards were found in the literature.

Lead Sulfate

Lead sulfate (syn. ANGLISLITE; FLEISULFAT (GERMAN); C.I. 77630; C.I. PIGMENT WHITE 3; FAST WHITE FREEMANS WHITE LEAD; LEAD BOTTOMS; LEAD DROSS (DOT); LEAD SULFATE, solid, containing more than 3% free acid [DOT]; MILK WHITE; MULHOUSE WHITE; SULFATE de PLOMB (FRENCH); SULFURIC ACID, LEAD [2+] SALT [1:1]), has the CAS number of 7446-14-2. Lead sulfate is used in storage batteries, paint pigments, lithography, preparing rapidly drying oil varnishes and weighting fabrics. Occupational exposure to lead sulfate dust may occur during its production and use. Workers at mines, ore processing facilities, and smelting and refining operations may be exposed to lead sulfate in air and solid since other lead compounds may be converted to the sulfate in these compartments (3).

Information pertaining to lead sulfate has been obtained from HSDB (3), RTECS (6), and Sax (7). Research on lead sulfate is limited, however its effects on human health are reasonably well-established. The sources searched contained no information on toxicity to plants and wildlife. Information available from the above-referenced sources includes various LDLos, OSHA PEL, ACGIH TLV, etc. (see Appendices).

Lead sulfate causes acute poisoning by ingestion. It also causes burning abdominal pain, nausea, vomiting, diarrhea, and less often, constipation. Among the other symptoms of lead sulfate poisoning are shock, weakness, pain and cramps.

Sax provides summary data including the following: Poison by intraperitoneal route. Moderately toxic by ingestion. Human mutagenic data. A corrosive irritant to skin, eyes and mucous membranes. When heated to decomposition, it emits very toxic fumes of lead and Sox.

Reference standards found in the literature included the following:
Human Carcinogenicity Group: B2

Mercury

Mercury (syn. COLLOIDAL MERCURY; KWIK [DUTCH] MERCURE [FRENCH]; MERCURIO [ITALIAN]; MERCURY, METALLIC [DOT]; NCI-C60399; QUECKSILBER [GERMAN]; QUICK SILVER; RCRA WASTE NUMBER U151; RTEC [POLISH]), has a CAS number of 7439-97-6. Mercury is naturally occurring, and mercury ore is found in rocks of all classes. It also occurs in fossil fuels and is released into the environment from volcanoes and hot springs. Mercury is used in barometers, thermometers, hydrometers; in mercury arc lamps producing ultraviolet rays, in switches, fluorescent lamps; in mercury boilers and mirrors. It is also a component of batteries and has laboratory uses (3).

Information pertaining to mercury has been obtained from ATSDR (1k), HEAST (2), HSDB (3), IRIS (4), RTECS (6), Sax (7), and EPA (9). Mercury has been well researched and its toxicity to humans and other species comparatively well-established. Information available from the above-referenced databases includes various LCLos, TCLos, TDLos, OSHA PEL, and ACGIH TLV (see Appendices).

Among the health effects of mercury ingestion are burning pain, sense of constriction, vomiting, severe purging, rapid weak pulse, collapse and death. Acute intoxication from inhaling mercury vapor in high concentrations used to be common among those who extracted mercury from its ores. The most consistent and pronounced effects of chronic exposure to elemental mercury vapor are neurological and psychiatric. Common symptoms include depression, irritability, exaggerated response to stimulation, excessive shyness, insomnia, emotional instability, forgetfulness, confusion. Excessive perspiration, uncontrolled blushing and tremors are also common. Mercury and its compounds occur naturally in trace amounts in plants growing in soils with low mercury concentrations. Numerous studies of fish/seafood concentrations have been reported for shrimp, flounder, clams, crabs, lobsters, salmon, oysters, scallops, trout, bass, catfish, sardines, pike, snapper and others. Concentrations have also been reported in birds and other animals (3). EPA reports toxicity data on both freshwater and saltwater plants and animals (9).

Sax provides summary data including the following: Poison by inhalation. An experimental tumorigen. Corrosive to skin, eyes and mucous membranes. Human systemic effects by inhalation: wakefulness, muscle weakness, anorexia, headache, diarrhea, liver changes, dermatitis, fever. An experimental teratogen. Experimental reproductive effects. Human mutagenic data. Explosive, reactive and incompatible with certain substances. When heated to decomposition, it emits toxic fumes of mercury.

Reference standards found in the literature included the following:
Human Carcinogenicity Group: D
Drinking Water Standard (MCL): 0.002 mg/L
Water Quality Criteria - freshwater: 4-day ave. conc. not to exceed 0.012 ug/L on ave. and 1-hour ave. conc. not to exceed 2.4 ug/L more than once every 3 years
- saltwater: 4-day ave. conc. not to exceed 0.025 ug/L on ave. and 1-hour ave. conc. not to exceed 2.1 ug/L more than once every 3 years.

Nickel

Nickel (syn. C.I. 77775; Ni 270; NICKEL 270; NICKEL DUST; NICKEL PARTICLES; NICKEL SPONGE; Ni 0901-S; Ni 4303T; NP 2; RANEY ALLOY; RANEY NICKEL), has the CAS number of 7440-02-0. Nickel is naturally occurring in the earth's crust. Natural sources of airborne particles that contain nickel include soil, sea, volcanoes, forest fires, and vegetation. Nickel is used for nickel plating; for various alloys; for coins, electrotypes, lightning rod tips, electrical contacts and electrodes, spark plugs and machinery parts; and in alloys for electronic and space applications. It is also used extensively for making stainless steel and other corrosion resistant alloys (3).

Information concerning nickel has been obtained from ATSDR (1l), HEAST (2), HSDB (3), IRIS (4), RTECS (6), Sax (7), and EPA (9). Nickel has been well researched and its toxicity toward humans and other species comparatively well-established. Information available from the above-referenced databases includes various LD50s, LDLos, Tds, TDLos, OSHA PEL, ACGIH TLV, etc. (see Appendices).

Acute toxicity from nickel inhalation includes headache, sore throat and hoarseness. Early symptoms after inhalation are dizziness, giddiness, and weakness. Large doses taken orally may cause nausea, vomiting, and diarrhea. Changes in the kidneys have also been observed. Nickel contact dermatitis is the most common reaction to nickel. Occupations at risk for nickel dermatitis included metal workers, electroplaters, hairdressers, nurses, and a host of others. Nickel carbonyl produced increases in the number of cases of nasal and lung cancer in nickel workers. No data on concentrations in plants, fish/seafood, or animals were reported (3). EPA reports acute and chronic toxicity to fish and invertebrates and states that both are spread throughout the range of sensitivity (9).

Sax provides summary data on nickel including the following: Poison by ingestion, intratracheal, intraperitoneal, subcutaneous and intravenous routes. An experimental carcinogen, neoplastigen, tumorigen, and teratogen. Experimental reproductive effects. Ingestion of soluble salts causes nausea, vomiting, diarrhea. Mutagenic data. Hypersensitivity to nickel is common and can cause allergic contact dermatitis, pulmonary asthma, conjunctivitis, and inflammatory reactions around nickel-containing medical implants and prostheses. Powders may ignite spontaneously in air. Nickel reacts violently with certain chemicals. Nickel catalysts have caused many industrial accidents.

Reference standards found in the literature included the following:
Human Carcinogenicity Group: A
Drinking Water Standard (MCL): 0.1 mg/L
Water Quality Criteria - freshwater: 4-day ave. conc. not to exceed e(0.8460[1n(hardness)]+1.1645)ug/L on ave. and 1-hour ave. conc. not to exceed e(0.8460[1n(hardness)]+3.3612)ug/L on ave. more than once every 3 years
- saltwater: 4-day ave. conc. not to exceed 8.3 ug/L on ave. and 1-hour conc. not to exceed 75 ug/L more than once every 3 years.

Silver

Silver (syn. ARGENTUM; C.I. 77829; SHELL SILVER; SILBER[GERMAN]; SILVER ATOM), has a CAS number of 7440-22-4. Silver occurs naturally in the earth's crust, and in both seawater and freshwater. Sources of silver in the atmosphere include emissions from smelting operations, cloud seeding (silver iodide), coal combustion, iron and steel production, cement manufacture, urban refuse incineration and cigarette tobacco. Silver is used in jewelry, for electroplating, as an ingredient in dental alloys, for food and beverage vessels and in aircraft engines. It is also a component of photographic materials and is used in coinage, mirrors and ceramic paints (3).

Information pertaining to silver has been obtained from ATSDR (1n), HEAST (2), HSDB (3), IRIS (4), RTECS (6), Sax (7), and EPA (9). Silver has been well researched and its toxicity toward humans and other species comparatively well-established. Information available from the above-referenced sources includes various Tds, TDLos, TCLos, OSHA PEL and ACGIH TLV (see Appendices).

Because many silver compounds dissolve in water and do not evaporate, the most common way that silver may enter the body of a person near a hazardous waste site is by drinking water that contains silver or eating food grown near the site in soil that contains silver. Silver compounds can cause areas of the skin and other body tissues to turn gray or blue-gray. However, the condition is thought to be only a cosmetic problem. Exposure to dust containing relatively high levels of silver compounds such as silver nitrate or silver oxide may cause breathing problems, lung and throat irritation and stomach pain. No studies of cancer or birth defects in animals from eating, drinking or breathing in silver compounds were found. Tests in animals show that silver compounds are likely to be life-threatening for humans only when large amounts (that is, grams) are swallowed and that skin contact with silver compounds is very unlikely to be life-threatening. Some water treatment methods use a form of silver to kill bacteria (1n). Fish/seafood and animal concentrations have been reported (3).

Sax provides summary data on silver including the following: An experimental tumorigen. Human systemic effects by inhalation: skin effects. Inhalation of dusts can cause argyrosis. Flammable in the form of dust when exposed to flame or by chemical reaction with various substances.

Reference standards found in the literature included the following:
Human Carcinogenicity Group: D
Drinking Water Standard (SMCL): 0.1 mg/L
Water Quality Criteria - freshwater: 4-day ave. conc. not to exceed e(1.72[1n(hardness)]-6.52) at any given time
- saltwater: conc. of total recoverable silver should not exceed 2.3 ug/L at any time.

Sodium cyanide

Sodium cyanide (syn. CYANOGRAN; CYMAG; HYDROCYANIC ACID, SODIUM SALT; RCRA WASTE NUMBER P106; etc.) has the CAS number of 143-33-9. It has a variety of uses including as a fumigant, a rodenticide, in cleaning metals, in the manufacture of dyes and pigments, as a chelating compound, as a component of salts for case hardening of steel, and as an agent for extraction of gold and silver from ores (4). It is manufactured in a number of ways, including by heating sodium amide with carbon, or by melting sodium chloride and calcium cyanamide together in an electric furnace (3). Pure sodium cyanide exists as a white solid in the form of granules, flakes or eggs. It is odorless when perfectly dry, but emits the odor of hydrogen cyanide when damp (4). It is very water soluble.

Information pertaining to sodium cyanide has been obtained from the IRIS and Sax (7). The chemical has been very well researched and its toxicity toward humans and other mammals is well established. The IRIS reports that sodium cyanide is highly toxic, with a probable lethal oral dose to humans of less than seven drops for a 150 pound person. It may be fatal if inhaled, swallowed, or absorbed through the skin. Other reported effects include burns to the skin and eyes. IRIS reports that EPA has set a level of 200 ug/L as a safe level for freshwater fish consumption. Sodium cyanide remains unclassified regarding carcinogenicity due to a lack of data. The proposed Maximum Contaminant Level (MCL) required under the Safe Drinking Water Act is 200 ug/L. The Ambient Water Quality Criteria for Human Health for water and fish is also 200 ug/L (4). The Ambient Water Quality Criteria for Aquatic Organisms is 22 ug/L (acute freshwater) and 5.2 ug/L (chronic freshwater), and 1.0 ug/L (acute marine) and none (chronic marine).

Sax summarizes the data as: A deadly human poison by ingestion and possibly other routes. A deadly experimental poison by ingestion, intraperitoneal, subcutaneous, intravenous, parenteral, intramuscular and ocular routes. An experimental teratogen. Human systemic effects by ingestion: hallucinations, distorted perceptions, muscle weakness and gastritis. Experimental reproductive effects. Flammable by chemical reaction with heat, moisture, acid. Many cyanides evolve hydrocyanic acid rather easily. This is a highly flammable gas and is highly toxic (7).

Sodium Dichromate

Sodium Dichromate (syn. BICHROMATE OF SODA; SODIUM BICHROMATE; CHROMIUM SODIUM OXIDE; WOLMAN SALTS CCA-TYPE B; CELCURE WOOD PRESERVATIVE; etc.) has the CAS number of 10588-01-9. It has a number of uses including as an oxidizing agent in the manufacture of dyes and inks; in the bleaching of non-edible fats, oils, and resins; in petroleum refining; for hardening gelatin; in the defoliation of cotton plants and other plants and shrubs; and as a wood preservative (3). It serves as a depressant in the process of ore flotation. It is manufactured primarily from sodium chromate and sulfuric acid (3).The chemical consists of bright red orange monoclinic, odorless crystals. A 10% aqueous solution has a pH of 3.5. It is very soluble in water and somewhat soluble in methanol (3).

Information pertaining to sodium dichromate has been obtained from the HSDB (3), RTECS (6), and Sax (7). The chemical has been well researched and its toxicity toward humans and other mammals is well established. Reported toxic effects range from severe skin and corneal burns (with dermal contact), to gastrointestinal corrosion and acute multisystem shock, renal failure, and sometimes death (with oral ingestion) (3). Sodium dichromate is a group 1 carcinogen, with sufficient evidence to indicate that the chemical is carcinogenic to both humans and animals (3). It has been shown to be an animal mutagen (3). The HSDB reports that chromium inhibits the uptake and translocation of essential elements in soybeans (toxic threshold of 1-5 ppm). It reports that the greatest risk of chromium toxicity to plants is in acidic, sandy soil having low organic content (3). RTECS reports the results of a number of LD50, LC50, and TDLo investigations (see Appendices).

Sax summarizes the data as: Poison by ingestion, skin contact, intravenous, intraperitoneal, and subcutaneous routes. An experimental tumorigen. Chromate salts are considered to be carcinogens. Human mutagenic data. A caustic and irritant. A powerful oxidizer. When heated to decomposition it emits toxic fumes of Na_2O. Violent reaction upon contact with a number of compounds (7).

No reference standards were found in the literature.

Sodium ferrocyanide

Sodium ferrocyanide (syn. SODIUM PRUSSIATE YELLOW; SODIUM HEXACYANOFERRATE; YELLOW PRUSSIATE OF SODA; TETRASODIUM FERROCYANIDE; etc.) has the CAS number of 13601-19-9. It has a variety of uses including in photography for bleaching, toning and fixing; to prevent caking of rock salt; as an additive to pickling paths; as a peptizing agent in rubber; as a food additive permitted in feed and drinking water of animals; for treatment of food-producing animals; permitted in human food consumption; and in mineral ore flotation. Information on its manufacture was not available in the resources checked. Pure sodium ferrocyanide exists as an odorless yellow solid. It is very water soluble.

Information pertaining to sodium ferrocyanide has been obtained from the HSDB (3). The literature reports that sodium ferrocyanide is much less toxic than sodium cyanide. The HSDB reports that the compound is moderately toxic, with a probable oral lethal dose of between one ounce and one pint for a 150 pound person (3). The HSDB reports that several grams of sodium ferrocyanide have been swallowed with no apparent ill effects. Specific data regarding other exposure hazards was not available. However, HSDB reports that waste ferrocyanides in streams and lakes should not exceed two ppm because irradiated solution becomes toxic to fish (3).

No Sax summary was available for sodium ferrocyanide.

No reference standards were found in the literature.

Sodium hydrosulfide

Sodium hydrosulfide (syn. HYDROGEN SODIUM SULFIDE; SODIUM BISULFIDE; SODIUM MERCAPTAN; etc.) has the CAS number of 16721-80-5. It has a variety of uses including in the production of heavy water for nuclear reactors; as a pulping agent in the production of paper; in dehairing hides; and as a flotation agent in ore processing (3). It is manufactured by the reaction of hydrogen sulfide with sodium hydroxide. Pure sodium hydrosulfide exists as white to colorless crystals. It is odorless when perfectly dry, but emits the odor of hydrogen sulfide when damp. It is very water soluble, and is also soluble in water and ether (3).

Information pertaining to sodium hydrosulfide has been obtained from the HSDB (3) and RTECS (6). The chemical has been well researched and its toxicity toward humans and other mammals is well established. The HSDB reports that sodium hydrosulfide is an alkaline corrosive. Severe burns are a common result of ingestion of or skin contact with concentrated solutions of the chemical. It is also an eye irritant. Pulmonary edema may result following inhalation of sodium hydrosulfide vapors. Under some conditions, extremely toxic hydrogen sulfide gas may be produced from sodium hydrosulfide (lethal inhalation exposure 600 ppm). RTECS indicates that sodium hydrosulfide may have mutagenic characteristics, although no sufficient data were provided (6). No information concerning the chemical's toxicity toward aquatic environments was found in the literature. No information concerning the effects to birds, plants, or other forms of life was found in the literature.

No Sax summary was available for sodium hydrosulfide.

No reference standards were found in the literature.

Sodium Hypochlorite

Sodium hypochlorite (syn. ANTIFORMIN; B-K LIQUID; CHLOROS; CLOROX; DAKINS SOLUTION; etc.) has the CAS number of 7681-52-9. It has a great variety of uses, including as a bleaching agent, as a disinfectant, germicide, deodorizer, and as a dye oxidizer (3). It is also serves as a depressant in ore flotation (8). It is manufactured in great quantity in the United States, primarily by combining chlorinated lime, anhydrous sodium carbonate, and sodium bicarbonate (3). It is a pale greenish-yellow liquid with a disagreeable, sweetish odor (3). Sodium hypochlorite crystals are very soluble in water. The liquid mixture thus produced is extremely alkaline (high pH).

Information pertaining to sodium hypochlorite has been obtained from the HSDB (3), RTECS (6), and Sax (7). The chemical has been very well researched and its toxicity toward humans and other mammals is well established. Reported toxic effects range from mild skin burns to death upon ingestion. A single study reported that a single application of sodium hypochlorite to the leaves of foliage plants caused severe necrosis, chlorosis, and leaf abscission (3). RTECS reports that there is insufficient data to classify the chemical as to its carcinogenicity toward humans or other animals. Little additional information concerning toxicity toward aquatic species, birds, or plants could be found. Information available from the above-referenced databases includes various LD50s and TDLos (see Appendices).

Sax summarizes the data as: Human mutagenic data. An eye irritant. Corrosive and irritating by ingestion and inhalation. The anhydrous salt is highly explosive and sensitive to heat or friction. Explosive reaction with formic acid (@ 55°C);phenylacetonitrile. Violent reaction upon contact with a number of compounds (7).

No reference standards were found in the literature.

Sodium Silicate

Sodium silicate (syn. SODIUM METASILICATE; CRYSTAMET; DISODIUM METASILICATE; DISODIUM MONOSILICATE; ORTHOSIL; SIMET A; METSO BEADS; DRYMET; etc.), has the CAS number of 6834-92-0. It is used in fireproofing mixtures and boiler compound; in laundry, dairy, and metal cleaning; as an additive in soaps and synthetic detergents; as a bleaching agent; and in mineral ore processing (3). It is obtained by the fusion of sodium carbonate with silicon dioxide or silica sand. It occurs as dustless white granules. It is soluble in water, insoluble in alcohol and acids or salt solutions, and insoluble in potassium and sodium salts (3).

Information pertaining to wood creosote has been obtained from the HSDB (3), RTECS (6), and Sax (7). Information available (see Appendices) from the above-referenced data bases includes TDLos, LDLos, and LD50 studies for mammals only. HSDB provided general information on the health effects of alkaline corrosives only, not on sodium silicate specifically. General problems associated with alkaline corrosives include severe burns (both skin and oral), and severe eye irritation. Inhalation of vaporized caustics may cause pulmonary edema.

Sax summarizes the data as: Poison by ingestion and intraperitoneal routes. A caustic material which is a severe eye, skin and mucous membrane irritant. Experimental reproductive effects. Ingestion causes gastrointestinal tract upset. Violent reaction with fluorine gas. When heated to decomposition it emits toxic fumes of Na2O.

No reference standards were found in the literature.

Sodium Sulfide

Sodium sulfide (syn. SODIUM MONOSULFIDE; SODIUM SULPHIDE; DISODIUM SULFIDE; etc.), has the CAS number of 1313-82-2. It has a variety of uses, including in the manufacture of rubber; in engraving; in cotton printing; as a depilatory agent in leather industries; in the preparation of pulp, paper, rayon, and cellophane film; in the production of heavy water for nuclear reactors; and as a solvent for gold mining (3). It is prepared commercially from elements in liquid ammonia. It exists in a variety of forms, including clear to yellow-pink to white crystals, yellow or brick-red lumps or flakes with a rotten egg odor (3). It is soluble in water and inorganic acids, slightly soluble in alcohol, and insoluble in most organic solvents.

Information pertaining to sodium sulfide has been obtained from the HSDB (3), RTECS (6), and Sax (7). Information available (see appendices) from the above-referenced databases includes several LD50 studies. Sodium sulfide produces a very caustic alkaline solution which can produce severe burns upon contact with skin, eyes, and mucous membranes. The material is very toxic; ingestion of small amounts (between one teaspoon and one ounce for a 150 pound person) can cause death. Sodium sulfide produces very toxic and flammable hydrogen sulfide upon addition to acids (3). No information concerning toxicity toward aquatic species, birds, or plants could be found.

Sax summarizes the data as: Flammable when exposed to heat or flame. Unstable and can explode on rapid heating or percussion. Reacts violently with carbon and a number of other materials. When heated to decomposition it emits toxic fumes of SO_x and Na_2O (7).

No reference standards were found in the literature.

Sulfur Dioxide

Sulfur dioxide (syn. BISULFITE; FERMENICIDE LIQUID; SCHWEFELDIOXYD [GERMAN]; SIARKI SWUTLENEK [POLISH]; SULFUROUS ACID ANHYDRIDE; SULFUROUS ANHYDRIDE; SULFUROUS OXIDE; SULFUR OXIDE; SULPHUR DIOXIDE, LIQUEFIED [DOT]), has a CAS number of 7446-09-5. Sulfur dioxide is naturally occurring as emissions from volcanoes. Hydrogen sulfide, from the natural decay of vegetation is probably oxidized to sulfur dioxide within hours. On a global basis, fossil fuel combustion accounts for 75 to 85% of man-made sulfur dioxide emissions, and industrial processes such as refining and smelting account for the remainder. It has been estimated by the Department of Labor that approximately 600,000 American workers may be occupationally exposed to sulfur dioxide. Some of the highest exposures occur when it is a by product, as in the metal smelting industry, and in the processing or combustion of high sulfur coal or oil. Other exposures occur in manufacture of sulfuric acid, fumigation, food preservation, wine making, and bleaching of many substances (3).

Information pertaining to sulfur dioxide has been obtained from HSDB (3), RTECS (6), and Sax (7). Sulfur dioxide has been well researched and its toxicity toward humans and other species comparatively well-established. Information available from the above-referenced databases includes various TCLos, LCLos, OSHA PEL, and ACGIH TLV (see Appendices).

Sulfur dioxide is extremely irritating to the respiratory tract. Coughing, sneezing, and swelling of the upper airway may occur. Serious eye injury or dermal frostbite injury may occur after direct contact with liquified sulfur dioxide. Victims surviving a serious inhalation injury may develop reactive airway disease, obstructive and restrictive lung disease, or chronic bronchitis. Nausea, vomiting, and abdominal pain may be seen as a result of ingestion. Green plants are extremely sensitive to atmospheric sulfur dioxide. Reportedly, alfalfa, barley, cotton, and wheat can be injured at relatively low doses. No data were provided on fish/seafood or animal concentrations (3).

Sax provides summary data including the following: A poison gas. Moderately toxic experimentally by inhalation. Mildly toxic to humans by inhalation. An experimental tumorigen and teratogen. Human systemic effects by inhalation: pulmonary vascular resistance, respiratory depression and other pulmonary changes. It chiefly affects the upper respiratory tract and the bronchi. It may cause edema of the lung, and can produce respiratory paralysis. Experimental reproductive effects. Human mutagenic data. A corrosive irritant to eyes, skin and mucous membranes. This material is so irritating that it provides its own warning of toxic concentration. Excessive exposures to high enough concentration of this material can be fatal. However, less than fatal concentration can be borne for fair periods of time with no apparent damage. It is a common air contaminant. It reacts violently with various substances. It will react with water or steam to produce toxic and corrosive fumes. It is incompatible with various substances. When heated to decomposition, it emits toxic fumes of SOx.

No reference standards were found in the literature.

Sulfonates

The group of chemical compounds known as sulfonates cannot be discussed as a group both because of the variability reported in their toxicities, and because of a lack of data.

Sulfuric Acid

Sulfuric acid (syn. ACIDE SULFURIQUE [FRENCH]; ACIDO SOLFORICO [ITALIAN]; BOV; DIPPING ACID; HYDROGEN SULFATE [DOT]; MATTING ACID; NORDHAUSEN ACID [DOT]; OIL OF VITRIOL; SCHWEFELSAEURELOESUNGEN [GERMAN]; SPENT SULFURIC ACID [DOT]; SULPHURIC ACID; VITRIOL BROWN OIL; VITRIOL, OIL OF [DOT]; ZWAVELZUUROPLOSSINGEN [DUTCH]) has a CAS number of 7664-93-9. Sulfuric acid is used in the manufacture of dyestuffs, other acids, parchment paper, glue and purification of petroleum; medication; electroplating baths; chemical intermediary for fertilizers; and dehydrating agent. Over a third of sulfuric acid is used in phosphate fertilizers. Sulfuric acid enters waters in a variety of ways: in accidental spills from railcar derailments, in wastewaters from mining properties where sulphides are a part of the ore or the rock being mined, in wastewaters from the steel industry, from the atmosphere, and as a decomposition product of effluents containing sulphur, thiosulphate, or other thionates. Sulfuric acid will ultimately react with calcium and magnesium in water to form sulfate salts (3).

Information pertaining to sulfuric acid has been obtained from HEAST (2), HSDB (3), RTECS (6), and Sax (7). Sulfuric acid has been well researched and its toxicity toward humans and other species comparatively well-established. Information available from the above-referenced databases includes various LC50s, LDLos, TCLos, OSHA PEL, and ACGIH TLV (see Appendices).

Sulfuric acid exotoxicity values are reported for mosquito fish, bluegill, flounder, shrimp and prawns. No data were reported for plant, fish/seafood or animal concentrations (3).

Sax provides summary data on sulfuric acid including the following: Human poison by unspecified route. Experimental poison by inhalation. Moderately toxic by ingestion. A severe eye irritant. Extremely irritating, corrosive, and toxic to tissue resulting in rapid destruction of tissue, causing severe burns. If much of the skin is involved, it is accompanied by shock, collapse and symptoms similar to those seen in severe burns. There are systemic effects secondary to tissue damage caused by contact with it. However, repeated contact with dilute solutions can cause a dermatitis, and repeated or prolonged inhalation of a mist of sulfuric acid can cause inflammation of the upper respiratory tract leading to chronic bronchitis. Sensitivity to sulfuric acid or mists or vapors varies with individuals. Workers exposed to low concentrations of the vapor gradually lose their sensitivity to its irritating action. Inhalation of concentrated vapors or mists can cause rapid loss of consciousness and serious damage to lung tissue. Severe exposure may cause a chemical pneumonitis; erosion of the teeth due to exposure to strong acid fumes has been recognized in industry. A common air contaminant. This is a very powerful, acidic oxidizer which can ignite or explode on contact with many materials. When heated it emits highly toxic fumes; will react with water or steam to produce heat; can react with oxidizing or reducing materials. When heated to decomposition, it emits toxic fumes of SOx.

No reference standards were found in the literature.

Zinc

Zinc (syn. BLUE POWDER; C.I. 77945; C.I. PIGMENT BLACK 16; C.I. PIGMENT METAL 6; EMANAY ZINC DUST; GRANULAR ZINC; JASAD; MERRILLITE; PASCO; ZINC DUST; ZINC POWDER; ZINC, POWDER OR DUST, NON-PYROPHORIC [DOT]; ZINC, POWDER OR DUST, PYROPHORIC [DOT], has a CAS number of 7440-66-6. Zinc is naturally occurring in several minerals. During blasting and crushing of ore, moderate losses of zinc to the atmosphere occur. During smelting, there are often large emissions. Zinc is used in bearings, electroplating, gold dental alloys, magnesium alloys, metallic driers, mixed metal stabilizers, in cadmium recovery and cooling towers (3).

Information pertaining to zinc has been obtained from ATSDR (1p), HSDB (3), IRIS (4), RTECS (6), Sax (7), and EPA (9). Zinc has been well researched and its toxicity toward humans and other species comparatively well-established. Information available from the above-referenced databases includes a TCLo, LD50s, and other indices (see Appendices).

Symptoms of zinc inhalation may start in 3 to 10 hours, and produce symptoms of flu which usually abate in 24 to 48 hours. Inhalation of soldering fume containing zinc chloride may cause asthma. Zinc salts produce gastritis ranging from a burning pain in the mouth and throat caused by zinc sulfate, to intense gastric pain, violent vomiting, diarrhea, shock, and possible death caused from zinc chloride, phosphide, and sulfate. Meat, fish, and poultry, as well as most crops contain zinc (3). EPA reports acute and chronic toxicities for numerous species of freshwater animals. The sensitivity range of freshwater plants to zinc is greater than that for animals. Zinc was also found to significantly bioaccumulate (9).

Sax provides summary data on zinc including the following: Human systemic effects by ingestion: cough, sweating. A human skin irritant. Pure zinc powder, dust, fume is relatively non-toxic to humans by inhalation. The difficulty arises from oxidation of zinc fumes immediately prior to inhalation or presence of impurities. Inhalation may cause sweet taste, throat dryness, cough, weakness, generalized aches, chills, fever, nausea, vomiting. Flammable in the form of dust when exposed to heat or flame. May ignite spontaneously in air when dry. Explosive in the form of dust when reacted with acids. Incompatible with various substances. When heated to decomposition, it emits toxic fumes.

Reference standards found in the literature included the following:
Human Carcinogenicity Group: D
Drinking Water Standard (SMCL): 5 mg/L
Water Quality Criteria - freshwater: 4-day ave. conc. not to exceed e(0.8473[1n(hardness)]+0.7613)ug/L on ave. and 1-hour ave. conc. not to exceed e(0.8473[1n(hardness)]+0.8604)ug/L more than once every 3 years
- saltwater: 4-day ave. conc. not to exceed 86 ug/L on ave. and 1-hour ave. conc. not to exceed 95 ug/L more than once every 3 years.

Zinc Sulfate

Zinc sulfate (syn. BONAZEN; BUFOPTO ZINC SULFATE; OP-THAL-ZIN; SULFATE de ZINC [FRENCH]; SULFURIC ACID, ZINC SALT [1:1]; VERAZINC WHITE COPPERAS; WHITE VITRIOL; ZINC SULPHATE; ZINC VITRIOL; ZINKOSITE, has a CAS number of 7733-02-0. Zinc, one of the most widely used micronutrients, is applied as sulfates (both basic and normal hydrates), carbonate, sulfide, phosphate, oxide, chelates, and other organic materials. It is also used in the production of rayon, as a feed supplement, and as a fertilizer ingredient. Other uses include herbicide treatments, soil treatments on lawns and in sewage against animal pathogenic bacteria (3).

Information pertaining to zinc sulfate has been obtained from HSDB (3), RTECS (6), and Sax (7). Zinc sulfate has been fairly well researched and its toxicity toward humans and other species reasonably well-established. Information available from the above-referenced databases includes various LD50s, LDLos, and TDLos (see Appendices).

The clinical effects of exposure to zinc sulfate (a zinc salt) are discussed with zinc. No plant, fish or seafood concentrations are reported (3).

Sax provides summary data for zinc sulfate including the following: Poison by intraperitoneal, subcutaneous and intravenous routes. Moderately toxic by ingestion. An experimental tumorigen and teratogen. Human systemic effects by ingestion: increased pulse rate without blood pressure decrease, blood pressure decrease, acute pulmonary edema, diarrhea and other gastrointestinal changes. Experimental reproductive effects.

No reference standards were found in the literature.

ORGANICS

XANTHATES

Xanthates

A number of the xanthates listed in the McNulty report (8) could not be found in the literature. These include:

Potassium sec amyl xanthate
Sodium amyl xanthate
Sodium isobutyl xanthate
Potassium hexyl xanthate
Amyl xanthate esters
Sodium disecbutyl xanthate
Xanthogen formates

Although their toxicities could not be researched directly, it is likely that they are similar to those found in the literature (see Sodium isopropyl xanthate, for example).

Potassium amyl xanthate

Potassium amyl xanthate (syn. AMYL POTASSIUM XANTHATE; XANTHIC ACID, PENTYL-, POTASSIUM SALT; CARBONODITHIOIC ACID, O-PENTYL ESTER, POTASSIUM SALT; AEROXANTHATE 350; etc.), has the CAS number of 2720-73-2 (6). It is used primarily in mineral flotation (8). McNulty reports that xanthates decompose to alkalis and organic sulfates, which do not contribute to acid mine drainage (8). Information concerning manufacture and physical characteristics was not available. Generally, xanthates are water soluble (7).

Limited information pertaining to potassium amyl xanthate has been obtained from RTECS (6). Unfortunately, the information available (see appendices) consisted only of a list of synonyms and one LD50 study with unreported results. No other toxicological data were available. However, information available from the databases of other xanthates may provide a reasonable approximation of the toxicological properties of potassium amyl xanthate. Using a worst case comparison (sodium isopropyl xanthate) indicates that the toxicity to fish may be quite high (2-13 ppm). The HSDB indicates that toxic effects in humans range from mild dermatitis to eye irritation. The estimated lethal dose for humans is quite low, between 1 teaspoon and 1 ounce for a 150 lb. person (3). RTECS indicates that some toxic effects are muscle contraction or spasticity; antipsychotic; and excitement (6). No information concerning toxicity toward birds, or plants could be found.

A Sax data summary was not available. However, Sax summarizes the data for sodium isopropyl xanthate as: Moderately toxic by parenteral route. A combustible

Potassium ethyl xanthate

Potassium ethyl xanthate (syn. ETHYL POTASSIUM XANTHATE; ETHYLXANTHIC ACID POTASSIUM SALT; CARBONODITHIOIC ACID, O-ETHYL ESTER, POTASSIUM SALT; etc.), has the CAS number of 140-89-6 (7). It is used primarily in mineral flotation (8). McNulty reports that xanthates decompose to alkalis and organic sulfates, which do not contribute to acid mine drainage (8). Information concerning manufacture was not available. It is a water soluble liquid.

Information pertaining to potassium ethyl xanthate has been obtained from RTECS (6) and Sax (7). Available information on the toxicology of potassium ethyl xanthate is limited. No information concerning toxicity toward aquatic species, birds, or plants could be found. Information available (see appendices) from the above-referenced databases includes various LD50s and TDLos. RTECS indicates that some toxic effects are muscle contraction or spasticity; antipsychotic; and excitement (6).

Sax summarizes the data as: Poison by subcutaneous and intravenous routes. Moderately toxic by ingestion. Combustible when exposed to heat or flame. Moderately explosive in the form of vapor when exposed to flame. Can react with oxidizing materials. When heated to decomposition it emits highly toxic fumes of K_2O and SO_x (7).

No reference standards were found in the literature.

Potassium Isobutyl Xanthate

(Note: Although McNulty (8) reports that sodium isobutyl xanthate is the constituent of concern only toxicological data concerning potassium isobutyl xanthate could be found. WCI believes that the toxicological characteristics of the two different salts are probably very similar).

Potassium isobutyl xanthate (syn. ISOBUTYL POTASSIUM XANTHATE; POTASSIUM ISOBUTYL XANTHOGENATE; CARBONODITHIOIC ACID-, o-(2-METHYLPROPYL) ESTER, POTASSIUM SALT; etc.) has the CAS number of 13001-46-2. It is used primarily as a promoter in mineral ore flotation (8). Information concerning its manufacture and physical characteristics was not available from the sources researched. Xanthates are generally soluble in water. McNulty reports that xanthates decompose to alkalis and organic sulfates, which do not contribute to acid mine drainage (8).

Limited information pertaining to potassium isobutyl xanthate has been obtained from RTECS (6) and Sax (7). Information available (see appendices) from the above-referenced databases consists of three LD50 studies only. RTECS indicates that some toxic effects are muscle contraction or spasticity; antipsychotic; and excitement (6).

Sax summarizes the data as: Poison by intravenous route. Moderately toxic by ingestion. When heated to decomposition it emits toxic fumes of SO_x and K_2O (7).

No reference standards were found in the literature.

Potassium Isopropyl Xanthate

Potassium isopropyl xanthate (syn. ISOPROPYLXANTHIC ACID, POTASSIUM SALT; CARBONIC ACID, DITHIO-, O-ISOPROPYL ESTER, POTASSIUM SALT; POTASSIUM ISOPROPYL XANTHOGENATE; etc.) has the CAS number of 140-92-1. It is used as a flotation reagent for base and precious metal ores (8). Information concerning its manufacture and physical properties was not available from the sources researched. McNulty reports that xanthates decompose to alkalis and organic sulfates, which do not contribute to acid mine drainage (8).

Limited information pertaining to potassium isopropyl xanthate has been obtained from RTECS (6). Unfortunately, the information available (see appendices) consisted only of a list of synonyms and one LD50 study. However, information available from the databases of other xanthates may provide a reasonable approximation of the toxicological properties of potassium isopropyl xanthate. Using a worst case comparison (sodium isopropyl xanthate) indicates that the toxicity to fish may be quite high (2-13 ppm). The HSDB indicates that toxic effects in humans range from mild dermatitis to eye irritation. The estimated lethal dose for humans is quite low, between 1 teaspoon and 1 ounce for a 150 lb. person (3). No information concerning toxicity toward birds, or plants could be found.

A Sax data summary was not available. However, Sax summarizes the data for sodium isopropyl xanthate as: Moderately toxic by parenteral route. A combustible material. When heated to decomposition it emits toxic fumes of SO_x and Na_2O (7).

No reference standards were found in the literature.

Potassium Secbutyl Xanthate

Potassium secbutyl xanthate (syn. CARBONIC ACID, DITHIO-, O-sec-BUTYL ESTER, POTASSIUM SALT; XANTHIC ACID, sec-BUTYL-, POTASSIUM SALT; etc.) has the CAS number of 141-96-8. It is used as a flotation reagent for base and precious metal ores (8). Information concerning its manufacture and physical properties was not available from the sources researched. McNulty reports that xanthates decompose to alkalis and organic sulfates, which do not contribute to acid mine drainage (8).

Limited information pertaining to potassium secbutyl xanthate has been obtained from RTECS (6). Unfortunately, the information available (see appendices) consisted only of a list of synonyms. No toxicological data was available. However, information available from the databases of other xanthates may provide a reasonable approximation of the toxicological properties of potassium secbutyl xanthate. Using a worst case comparison (sodium isopropyl xanthate) indicates that the toxicity to fish may be quite high (2-13 ppm). The HSDB indicates that toxic effects in humans range from mild dermatitis to eye irritation. The estimated lethal dose for humans is quite low, between 1 teaspoon and 1 ounce for a 150 lb. person (3). No information concerning toxicity toward birds, or plants could be found.

A Sax data summary was not available. However, Sax summarizes the data for sodium isopropyl xanthate as: Moderately toxic by parenteral route. A combustible material. When heated to decomposition it emits toxic fumes of SO_x and Na_2O.

No reference standards were found in the literature.

Sodium Ethylxanthate

Sodium ethylxanthate (syn. ETHYLXANTHIC ACID, SODIUM SALT; SODIUM XANTHOGENATE; etc.) has the CAS number of 140-90-9. It is used as a flotation reagent for base and precious metal ores (8). Information concerning its manufacture was not available from the sources researched. Sax reports that the material is a yellow powder, soluble in water and alcohol (7). McNulty reports that xanthates decompose to alkalis and organic sulfates, which do not contribute to acid mine drainage (8).

Limited information pertaining to sodium ethylxanthate has been obtained from Sax (7). Unfortunately, the information available (see appendices) consisted only of a list of synonyms and a description of physical properties. However, information available from the databases of other xanthates may provide a reasonable approximation of the toxicological properties of potassium isopropyl xanthate. Using a worst case comparison (sodium isopropyl xanthate) indicates that the toxicity to fish may be quite high (2-13 ppm). The HSDB indicates that toxic effects in humans range from mild dermatitis to eye irritation. The estimated lethal dose for humans is quite low, between 1 teaspoon and 1 ounce for a 150 lb. person (3). No information concerning toxicity toward birds, or plants could be found.

Sax summarizes the data as: Flammable when exposed to heat or flame. When heated to decomposition it emits toxic fumes of Na_2O and SO_x. Sax summarizes sodium isopropyl xanthate as: Moderately toxic by parenteral route. A combustible material. When heated to decomposition it emits toxic fumes of SO_x and Na_2O (7).

No reference standards were found in the literature.

Sodium Isopropyl Xanthate

Sodium isopropyl xanthate (syn. ISOPROPYLXANTHIC ACID, SODIUM SALT; PROXANSODIUM; AEROXANTHATE 343; etc.) has the CAS number of 140-93-2. It has several common uses, including as a chemical weed killer; as a fortifying agent for certain oils; and as a flotation reagent for base and precious metal ores (3). Information concerning its manufacture was not available from the sources researched. It exists normally as a white to yellowish powder with a slightly unpleasant odor (3). Sodium isopropyl xanthate is soluble in water. McNulty reports that xanthates decompose to alkalis and organic sulfates, which do not contribute to acid mine drainage (8).

Limited information pertaining to sodium isopropyl xanthate has been obtained from HSDB (3), RTECS (6) and Sax (7). Information available (see appendices) from the above-referenced databases includes one LDLo study and one LD50. The LD50 study indicated that the toxicity to fish may be quite high (2-13 ppm). The HSDB indicates that toxic effects in humans range from mild dermatitis to eye irritation. The estimated lethal dose for humans is quite low, between 1 teaspoon and 1 ounce for a 150 lb. person (3). No information concerning toxicity toward birds, or plants could be found.

Sax summarizes the data as: Moderately toxic by parenteral route. A combustible material. When heated to decomposition it emits toxic fumes of SO_x and Na_2O.

No reference standards were found in the literature.

ORGANICS

DITHIOPHOSPHATES (DTP)

Dithiophosphates

Two of the dithiophosphates listed in the McNulty report (8) could not be found in the literature. These are:

Sodium dialyl dithiophosphate
aryl dithiophosphate acid

Although their toxicities could not be researched directly, it is likely that they are similar to those found in the literature (see Sodium diisopropyl dithiophosphate, for example).

Sodium Diethyl Phosphorodithioate

Sodium diethyl phosphorodithioate (syn. SODIUM DITHIOPHOSPHATE; PHOSPHORIC ACID, O-O-DIETHYL ESTER, SODIUM SALT; SODIUM AEROFLOAT; etc.) has the CAS number of 3338-24-7. It is used in the synthesis of organophosphate insecticides and as a flotation agent for zinc and copper sulfides (3). It is produced by the reaction of ethyl alcohol and phosphorous pentasulfide, followed by the reaction of the resulting diethyl phosphorodithioate with sodium hydroxide (3). Information concerning its physical properties was not available from the sources researched. McNulty reports that dithiophosphates decompose to phosphates which do not contribute to acid mine drainage (8).

Limited information pertaining to sodium diethyl phosphorodithioate has been obtained from the HSDB (3) and RTECS (6). Reported adverse effects include moderate to severe burns upon oral ingestion; swelling of the eyes and blindness upon eye contact; and chest pain and pulmonary edema upon inhalation of fumes. A single LD50 study on rainbow trout indicates that it is not strongly toxic to them (LD50 400 ppm/96 hr). A mutagenicity study reported that the compound had no carcinogenic effects (3). No information concerning toxicity toward birds or plants could be found.

A Sax data summary was not available.

No reference standards were found in the literature.

Sodium di-sec-butyl phosphorodithioate

Sodium di-sec-butyl phosphorodithioate (syn. AEROFLAT 238; SODIUM DI-SECBUTYL DITHIOPHOSPHATE; PHOSPHORODITHIOIC ACID, O-O-DI-SEC-BUTYL ESTER, SODIUM SALT; etc.) has the CAS number of 3361992-0. It is used as a flotation promoter in the processing of gold, silver, copper, and zinc sulfide ores (3). It is produced by the reaction of secbutyl alcohol and phosphorous pentasulfide, followed by the reaction of the resulting disecbutyl phosphorodithioate with sodium hydroxide (3). Information concerning its physical properties was not available from the sources researched. McNulty reports that dithiophosphates decompose to phosphates which do not contribute to acid mine drainage (8).

Limited information pertaining to sodium disecbutyl phosphorodithioate has been obtained from the HSDB (3). Reported adverse effects include moderate to severe burns upon oral ingestion; swelling of the eyes and blindness upon eye contact; and chest pain and pulmonary edema upon inhalation of fumes. A single LD50 study on rainbow trout (for a similar dithiophosphate) indicates that it is not strongly toxic to them (LD50 400 ppm/96 hr). A mutagenicity study reported that the compound had no carcinogenic effects (3). No information concerning toxicity toward birds or plants could be found.

A Sax data summary was not available.

No reference standards were found in the literature.

Sodium diisopropyl phosphorodithioate

Sodium diisopropyl phosphorodithioate (syn. SODIUM DIISOPROPYL DITHIOPHOSPHATE; PHOSPHORODITHIOIC ACID, O-O-DIISOPROPYL ESTER, SODIUM SALT; etc.) has the CAS number of 27205-99-8. It is used as a flotation promoter in the processing of gold, silver, copper, and zinc sulfide ores (3). It is produced by the reaction of isopropyl alcohol and phosphorous pentasulfide, followed by the reaction of the resulting diisopropyl phosphorodithioate with sodium hydroxide (3). Information concerning its physical properties was not available from the sources researched. McNulty reports that dithiophosphates decompose to phosphates which do not contribute to acid mine drainage (8).

Limited information pertaining to sodium diisopropyl phosphorodithioate has been obtained from the HSDB (3). Reported adverse effects include moderate to severe burns upon oral ingestion; swelling of the eyes and blindness upon eye contact; and chest pain and pulmonary edema upon inhalation of fumes. A single LD50 study on rainbow trout (for a similar dithiophosphate) indicates that it is not strongly toxic to them (LD50 400 ppm/96 hr). A mutagenicity study reported that the compound had no carcinogenic effects (3). No information concerning toxicity toward birds or plants could be found.

A Sax data summary was not available.

No reference standards were found in the literature.

OTHER ORGANICS

Alcohols

The group of chemical compounds known as alcohols cannot be discussed as a group because of the high variability reported in their toxicities. For example, IRIS reports that propargyl alcohol (CAS 107-19-7) has a NOAEL (no observable adverse effects level) of 5 mg/kg-day, while methanol (CAS 67-65-1) has a NOAEL of 500 mg/kg-day. Phenol, technically an alcohol, and known to be highly toxic, has a NOAEL of 60 mg/kg-day. In addition, alcohols are produced in nature, and may be found in all natural systems.

alpha-Terpineol

Alpha-terpineol (syn. p-MENTH-1-EN-8-OL (8CI); 3-CYCLOHEXENE-1-METHANOL, ALPHA, ALPHA, 4-TRIMETHYL; etc.) has the CAS number of 98-55-5. It has a variety of uses including in the manufacture of perfumes; denaturing fats for soap manufacture; as an antiseptic; as a synthetic flavor body in chewing gum; as an antioxidant; as the major component in pine oil cleaner and disinfectant; and in ore flotation (3). It is manufactured by heating terpin hydrate with phosphoric acid and distilling; and by fractional distillation of pine oil (3). Pure alpha-terpineol is a white powder, although it usually is dissolved in alcohol and forms a clear liquid with a sweet lime or floral taste (5). Chewing gum flavored with the substance may contain 40 ppm of alpha-terpineol (3).

Information pertaining to alpha-terpineol has been obtained from the HSDB (3), RTECS (6), and Sax (7). The chemical has been very well researched and its toxicity toward humans and other mammals is well established. The HSDB reports that alpha-terpineol is moderately toxic, with a probable lethal oral dose to humans of between one ounce and one pint for a 150 pound person (3). Other reported effects range from lesions of the lips, mild respiratory depression, nausea, renal failure, and death to a child who ingested 1/2 ounce. The substance is reportedly nonmutagenic (3). HSDB reports that it may be released to the environment as a natural product; it is a volatile component of essential oils, nectarines, and cranberry juice. Human release to the environment includes waste streams from wood pulping industries, petrochemical companies and waste disposal sites. If released to the atmosphere, vapor-phase alpha-terpineol is expected to degrade by reaction with photochemically produced hydroxyl radicals (est. half life of four hours). Based on its relatively high water solubility, long distance transport in water systems may be significant. If released to the soil, it is expected to leach significantly. Biodegradation in soil may be moderately fast. A volatilization half-life of 19 days has been estimated for a river model. One biodegradation study observed 99% degradation in seven days. Studies indicate that it will not bioconcentrate significantly in aquatic organisms (3).

Sax summarizes the data as: Moderately toxic by intramuscular route. When heated to decomposition it emits acrid smoke and fumes (7).

No reference standards were found in the literature.

Citric Acid

Citric Acid (syn. CITRO; 2-HYDROXY-1,2,3-PROPANETRICARBOXYLIC ACID; BETA-HYDROXY-TRICARBOXYLIC ACID; etc.), has the CAS number of 77-92-9. It is used as a sequestering agent to remove trace metals; as an acidulant, pH regulator, flavor enhancer, preservative, and antioxidant in foods; in metal cleaning and finishing; as a water-conditioning agent; and as a silica depressant in mineral ore flotation (3, 8). It is produced from a variety of processes, including mold fermentation of sugar cane molasses or extraction from citrus fruits (3). In solid form it exists as colorless to white translucent odorless crystals. A citric acid solution has a pleasant, sour taste (3). It is insoluble in benzene, but very soluble in water (7).

Information pertaining to kerosene has been obtained from the HSDB (3), RTECS (6), and Sax (7). Information available (see appendices) from the above-referenced databases includes various LD50s and TDLos. The chemical has been very well researched and its toxicity toward humans and other mammals is well established. The HSDB reports that the most serious toxic effects following ingestion of highly concentrated solutions are nausea, vomiting, and diarrhea resulting from an irritation of the digestive tract. Upon inhalation of citric acid powder, the victim may exhibit coughing and wheezing. Direst dermal contact may burn the skin and eyes. No information concerning toxicity toward aquatic species, birds, or plants could be found.

Sax summarizes the data as: Poison by intravenous route. Moderately toxic by subcutaneous and interperitoneal routes. Mildly toxic by ingestion. A severe eye and moderate skin irritant. An irritating organic acid, some allergenic properties...(7).

No reference standards were found in the literature.

Dextrin

Dextrin (syn. artificial gum; dextran; starch gum; tapioca; vegetable gum; etc.) has the CAS number of 9004-53-9. It is used in the production of food and as a depressant in mineral ore flotation. It is an intermediate product formed by the hydrolysis of starches. It is one of a number of chemicals in the dextrin class of compounds. Dextrin compounds are usually a white or yellow powder or granules, soluble in water and insoluble in alcohol or ether. These compounds have colloidal properties (7).

Information pertaining to dextrin and related compounds has been obtained from the RTECS (6) and Sax (7). Although toxicity to humans is obviously low, no information concerning toxicity towards aquatic animals, plants, or birds could be found in the literature. Information from the above-referenced databases consists of one LD50 study (see Appendices).

Sax summarizes the data as: Mildly toxic by intravenous route. When heated to decomposition it emits acrid smoke and irritating flames (7).

No reference standards were found in the literature.

Glycols

The group of chemical compounds known as glycols cannot be discussed as a group both because of the variability reported in their toxicities, and because of a lack of data. Although IRIS contains partial data for 14 glycols, data on the reference doses for only three were reported. These ranged from 200 mg/kg-day for ethylene glycol (CAS 107-21-1) to 1000 mg/kg-day for butylphthalyl butylglycolate (CAS 85-70-1).

Guar Gum

Guar gum (syn. A-20D; CYAMOPSIS GUM; INDALCA AG; LYCOID DR; SUPERCOL U POWDER; etc.), has the CAS number of 9000-30-0. It has a variety of uses, including as a protective colloid, stabilizer, thickening and film forming agent for cheese, salad dressing, ice cream etc; in bulk laxatives and appetite depressants; and as a flocculent in mining (3). It is obtained from the seeds of the guar plant (*Cyamopsis tetragonolobus*). It is a yellowish white, free flowing powder, completely soluble in water. It is practically insoluble in oils, greases, hydrocarbons, ketones, and esters (3). Water solutions are tasteless and odorless, with a pale, translucent gray color, and a neutral pH.

Information pertaining to guar gum has been obtained from the HSDB (3), RTECS (6), and Sax (7). The chemical has been very well researched and its toxicity toward humans and other mammals is well established. No information concerning toxicity toward aquatic species, birds, or plants could be found. Information available from the above-referenced databases includes various LD50s and TDLos (see Appendices).

Sax summarizes the data as: Mildly toxic by ingestion. A stabilizer used as a food additive. When heated to decomposition it emits acrid smoke and irritating fumes (7). The HSDB reports that normal exposures to guar gum would not be expected to produce toxicity (3). There is no evidence of any carcinogenic effects (6,7).

No reference standards were found in the literature.

Linoleic Acid

Linoleic acid (syn. CIS-9, CIS-12-OCTADECADIENOIC ACID; 9,12-LINOLEIC ACID; Emersol 310; GRAPE SEED OIL; UNIFAC 6550; LINOLIC ACID; POLYLIN NO 515; etc.), has the CAS number of 60-33-3. It has a variety of uses, including in the manufacture of paints, coatings, emulsifiers, and vitamins; in soaps medicines, and feed; and in ore flotation. It is a common component of many vegetable oils and therefore of most normal diets (3). It is made primarily from the hydrolysis of various animal and vegetable fats and oils. It is a colorless to straw-colored. It is practically insoluble in water, but very soluble in organic solvents (3).

Information pertaining to linoleic acid has been obtained from the HSDB (3), RTECS (6), and Sax (7). The chemical has been very well researched and its toxicity toward humans and other mammals is well established. No information concerning toxicity toward aquatic species, birds, or plants could be found. Information available (see appendices) available from the above-referenced databases includes various LD50s and TDLos.

Sax summarizes the data as: A human skin irritant. Ingestion can cause nausea and vomiting. A nutrient and/or dietary supplement food additive. When heated to decomposition it emits acrid smoke and irritating fumes. Oxidized linoleic acid shows experimental reproductive effects. Linoleic acid mixed with oleic acid is an experimental tumorigen (7). The HSDB reports a TXHR=1.1 (practically nontoxic:probable oral lethal dose for humans is above 15 g/kg, more than one quart, or 2.2 pounds, for a 150 pound person) (3). Oleic acid was found to be negative when tested for mutagenicity using the Salmonella/microsome preincubation assay, applying the standard protocol approved by the National Toxicology Program (NTP) (3).

No reference standards were found in the literature.

Isobutylmethylcarbinol

Isobutylmethylcarbinol (syn. MAOH; METHYL AMYL ALCOHOL; 2-METHYL-4-PENTANOL;MIBC; etc.), has the CAS number of 108-11-2. It has a variety of uses, including as a solvent for dyestuffs, oils, gums, resins, waxes, and nitrocellulose, in the manufacture of lubricant additives, solvents, and plasticizers, and in ore froth flotation (3). It is prepared commercially by reduction of mesityl oxide in an acetic acid/platinum catalyst solution (3). It is a colorless liquid, soluble in alcohol and ether, and is miscible with most common organic solvents.

Information pertaining to isobutylmethylcarbinol has been obtained from the HSDB (3), RTECS (6), and Sax (7). The chemical has been moderately researched and its toxicity toward humans and other animals generally established. No information concerning toxicity toward aquatic species, birds, or plants could be found. Information available from the above-referenced databases includes various LD50s, TDLos, OSHA PELs, ACGIH TLVs, and upper and lower explosive limits (see Appendices). Exposure through inhalation has been the most widely investigated route. The HSDB reports that exposure to higher alcohols, in general, results primarily in depression of the central nervous system, hypotension, nausea, vomiting, and diarrhea (3).

The HSDB reports that isobutylmethylcarbinol released to the environment will be expected to exhibit high to very high mobility in soil. It will be expected to rapidly volatilize from surface waters and will biodegrade in soils. It is not expected to adsorb to sediment or to bioaccumulate in aquatic organisms. It has an expected half-life of 23 hours.

Sax summarizes the data as: Moderately toxic by ingestion, skin contact and intraperitoneal routes. Mildly toxic by inhalation. A skin and severe eye irritant. Inhalation of high concentrations can cause anesthesia. Flammable when exposed to heat or flame; can react with oxidizing materials. When heated to decomposition it emits acrid smoke and fumes (7).

No reference standards were found in the literature.

2-Mercaptobenzothiazole

2-Mercaptobenzothiazole (syn. MBT; ROKON; SULFADENE; USAF GY-3; PENNAC MBT POWDER; etc.), has the CAS number of 149-30-4. It has a variety of uses, including as a fungicide, bactericide, wood preservative, and in ore flotation (6). Information on its manufacture was not available. It is a light yellow powder (7).

Information pertaining to 2-mercaptobenzothiazole has been obtained from RTECS (6) and Sax (7). The chemical has been well researched and its toxicity toward humans and other mammals is well established. No information concerning toxicity toward aquatic species, birds, or plants could be found. Information available (see appendices) from the above-referenced databases includes various LD50s and TDLos.

Sax summarizes the data as: Poison by intraperitoneal routes. Moderately toxic by ingestion. An experimental tumorigen, teratogen and carcinogen. Other experimental reproductive effects. Incompatible with oxidizers. When heated to decomposition or on contact with acids or acid fumes it emits toxic SOx and NOx (7).

No reference standards were found in the literature.

Oleic Acid

Oleic acid (syn. CIS-9-OCTADECENOIC ACID; ELAIC ACID; EMERSOL 210; GROCO 2; NEO-FAT 90-04; PAMOLYN 100; VOPCOLENE 27; etc.), has the CAS number of 112-80-1. It has a variety of uses, including wool oiling, the manufacture of soaps, rodent extermination (barium oleate), in hand creams, in medicine, and in ore flotation. It is made primarily from the hydrolysis of various animal and vegetable fats and oils. It is a yellowish, oily liquid with a peculiar lard-like odor and taste. It is practically insoluble in water, but very soluble in organic solvents (3).

Information pertaining to oleic acid has been obtained from the HSDB (3), RTECS (6), and Sax (7). The chemical has been very well researched and its toxicity toward humans and other mammals is well established. The HSDB reports a TXHR=1.1 (practically nontoxic:probable oral lethal dose for humans is above 15 g/kg, more than one quart, or 2.2 pounds, for a 150 pound person) (3). Oleic acid was found to be negative when tested for mutagenicity using the Salmonella/microsome preincubation assay, applying the standard protocol approved by the National Toxicology Program (NTP) (3). No information concerning toxicity toward aquatic species, birds, or plants could be found. Information available (see appendices) available from the above-referenced databases includes various LD50s and TDLos.

Sax summarizes the data as: Poison by intravenous route. Mildly toxic by ingestion. An experimental tumorigen. Mutagenic data. A human and experimental skin irritant (7).

No reference standards were found in the literature.

Dowfroth 250

Dowfroth 250 (syn. POLYPROPYLENE GLYCOL METHYL ETHER; UCON LB-1715; JEFFOX OL 2700; alpha-METHYL-omega-HYDROXYPOLY(OXY(METHYL-1-2-ETHANEDIYL));etc.) has the CAS number of 37286-64-9. It has several uses, including as a functional fluid and diluent for hydraulic fluids; as a water-insoluble lubricant; and as a flotation frother in mineral ore flotation (3). It is produced by the reacting propylene oxide with alcohol over a catalyst (3). It is an amber liquid, completely miscible with water.

Information pertaining to Dowfroth 250 has been obtained from the HSDB (3), RTECS (6), Sax (7), and from the Dow Material Safety Data Sheet (MSDS) (9). The chemical has not been well researched and its toxicity toward humans and other mammals is not well known. However, glycols in general have been extensively researched. Toxic effects of glycols range from ethanol-like inebriation, ataxia, and slurred speech to coma and death, depending on the quantity ingested (3). Dow reports that the chemical may cause moderate eye irritation, and that single dose toxicity is low (9). Death has occurred after the ingestion of as little as 30 ml yet survival has been reported after the ingestion of 2 liters (3). No additional information concerning toxicity toward aquatic species, birds, or plants could be found. Dow reports that no oral LD50 has been established (9). RTECS provides data on several LD50 studies (6).

Sax summarizes the data as: Mildly toxic by ingestion. When heated to decomposition it emits acrid smoke and irritating fumes (7).

No reference standards were found in the literature.

Diphenylthiourea

Diphenylthiourea (syn. DIPHENYLTHIOCARBAMIDE; 1,3-DIPHENYLTHIOUREA; RHENOCURE CA; STABILISATOR C; SULFOCARBANILIDE; USAF EK-245; VALKACIT; etc.), has the CAS number of 102-08-9. It has a variety of uses, including as a vulcanizing accelerator; in the production of synthetic organic pharmaceuticals; and as a flotation agent in mineral ore processing (3). Information on its manufacture was not found in the literature searched. It is a white to faint gray powder, almost insoluble in water, freely soluble in alcohol and ether, and very soluble in chloroform and olive oil (3). McNulty reports that the decomposition products of diphenylthiourea include cyclic organics (including benzene) and organic sulfates, with no acidity being generated (8).

Information pertaining to diphenylthiourea has been obtained from the HSDB (3), RTECS (6), and Sax (7). The chemical has not been well researched and its toxicity toward humans and other animals not well established. However, Hawley reports that the compound has low toxicity (*). No information concerning toxicity toward aquatic species, birds, or plants could be found. Information available from the above-referenced databases includes various LD50s and LDLos (see Appendices).

Sax summarizes the data as: Moderately toxic by ingestion and intraperitoneal routes. When heated to decomposition it emits highly toxic fumes of SOx and NO_x (7).

No reference standards were found in the literature.

Isopropyl Ether

Isopropyl ether (syn. DIISOPROPYL ETHER; 2-ISOPROPOXYPROPANE; etc.), has the CAS number of 108-20-3. It has a variety of uses, including as a solvent for animal, vegetable, mineral oils, waxes and resins; and as a solvent in mineral ore extraction and processing (3). It is obtained in large quantities as a by-product from the manufacture of isopropyl alcohol from propylene by the sulfuric acid process (3). It is a colorless liquid with a sweet, slightly sharp, pungent odor like camphor and ethyl ether (3). It is slightly soluble in water, soluble in acetone, and miscible with most organic solvents.

Information pertaining to calcium hydroxide has been obtained from the HSDB (3) ,RTECS (6) and Sax (7). Information available (see appendices) available from the above-referenced databases includes LD50 and LC50 studies. The fatal oral dose is estimated to be one to two ounces. Inhalation may result in dizziness, giddiness, euphoria, and central nervous system depression. Acute industrial exposure deaths are rare (3). Prolonged exposure to high vapor concentrations may cause eye injury. Ether is irritating to the mucous membranes. Isopropyl ether is not thought to be carcinogenic (3). Limited fish data are presented in the HSDB. The HSDB also reports that isopropyl ether released to soil will be subject to volatilization. It will be expected to exhibit moderate mobility in soil and , therefore, it may leach to groundwater. It will not be expected to hydrolyze in soil. If released to water, it will not be expected to significantly adsorb to sediment or suspended particulate matter, bioconcentrate in aquatic organisms, hydrolyze, or directly photolyze, based upon estimated physical-chemical properties or analogies to other structurally related aliphatic ethers. Isopropyl ether in surface water will be subject to rapid volatilization with estimated half-lives of 3.3 hours and 40 hours for volatilization from a river one meter deep flowing one meter/sec with a wind velocity of three meters/sec. It may be resistant to biodegradation in environmental media.

Sax summarizes the data as: Moderately toxic by intraperitoneal and possibly other routes. Mildly toxic by ingestion, inhalation, and skin contact. A skin irritant. A very dangerous fire and explosion hazard.

No reference standards were found in the literature.

NON-COMPOUND-SPECIFIC ORGANICS

AMNO, Black Powder, and Dynamite

AMNO (ammonium nitrate/fuel oil blasting agent, black powder, and dynamite are all explosive agents used in mineral ore exploration and extraction. None of the databases searched contained data concerning the toxicities of these compounds.

Coal Creosote

Coal creosote (syn. BRICK OIL; COAL TAR OIL; CRESOSTE OIL; TAR OIL; WASH OIL; etc.), has the CAS number of 8001-58-9. It has a variety of uses, including as a feedstock for the production of carbon black; in antifungal preparations; as a vermicide; as a wood preservative; waterproofing agent; animal dip; and as a frothing agent for mineral ore separation (3). It is obtained by distillation of coal and as a by-product of conventional coal coking. Some of the polynuclear aromatic hydrocarbons which have been found in coal creosote are anthracene, benz(A)anthracene, benzo(B)chrysene, benzo(J)fluoranthene, benzo(G,H,I)perlyene, chrysene, fluoranthene, phenanthrene, and pyrene (3). It is a black to brown oily liquid with an aromatic, smoky odor and a burning caustic taste (3). It is slightly soluble in water, miscible in alcohol and ether, and soluble in glycerine and dimethyl sulfate (3).

Information pertaining to coal creosote has been obtained from the HSDB (3), RTECS (6), and IRIS (4). Information available (see appendices) available from the above-referenced databases includes LD50 and LC50 studies for mammals, fish, and birds. Coal creosote is a Group 2A carcinogen (probable human carcinogen). It is also known to be a carcinogen to other mammals. Ingestion of greater than 5% solution may result in oral burns. Eye exposure may result in severe burns. Contact with the skin or mucous membranes may cause intense burning and itching and gangrene in isolated instances. Deaths from large doses of creosote appear to be largely due to cardiovascular collapse. Death has occurred following ingestion of 7 grams by adults and 1-2 grams by children. Acute toxicity is believed to be caused by the presence of phenol or phenolic compounds (3). Laboratory bioassays indicate a generally moderate toxicity to aquatic organisms.

Sax summarizes the data as: Moderately toxic by ingestion. Experimental reproductive effects. Mutagenic data. When heated to decomposition it emits acrid smoke and fumes.

No reference standards were found in the literature.

Coal tar

Coal tar (syn. COAL-TAR PITCH VOLATILES; PITCH; COAL-TAR PITCH) has the CAS number of 65996-93-2. Limited information concerning coal tar was found in RTECS (6) and Sax(7) only.

RTECS reports several TDLos, TLVs, and PELs, and indicates that there is sufficient evidence to classify coal tar as a human carcinogen (6).

Sax summarizes the data as: An experimental carcinogen and neoplastigen by skin contact. When heated to decomposition it emits acrid smoke and fumes (7).

No reference standards were found in the literature.

Crude Petroleum, Oils, Greases, Fuel Oil, Gasoline, Diesel Fuel

The compounds known as crude petroleum, oils, greases, fuel oil, gasoline, and diesel fuel are characterized as being mixtures of a large number of semi-specific hydrocarbons. Crude oil, oils, and greases generally contain a higher fraction of "heavy hydrocarbons", i.e., large, heavy, complex molecules (straight chain, branched, and aromatic). Fuel oil and diesel fuel generally arecomposed of medium-weight hydrocarbons, while gasoline is at the lighter end. It is not really possible or worthwhile to try to discuss each of these groups individually, since their toxicities are affected by the individual chemicals which make up the mixture. A general indication of the toxicity of hydrocarbons can be found by reading the discussion on the various hydrocarbons presented elsewhere in this paper (see kerosene; mineral oil; coal creosote; and coal tar).

Inorganic lubricants

The group of chemical compounds known as inorganic lubricants cannot be discussed as a group because it is a diverse group of chemicals with widely varying physical and chemical properties. WCI requires the specific names of the compounds of concern before their toxicities can be researched.

Kerosene

Kerosene (syn. COAL BASE; COAL OIL; DEOBASE; UN1233; etc.), has the CAS number of 8008-20-6. It is burned as fuel in a variety of items, including lamps, flares, and stoves; as a motor fuel; as heating fuel; as a degreaser and solvent; and as a promoter in mineral ore flotation. It is produced from the fractional distillation of petroleum. It is a pale yellow or water-white, mobile, oily liquid with a characteristic, not altogether disagreeable odor. It is insoluble in water, but miscible with other organic solvents (3).

Information pertaining to kerosene has been obtained from the HSDB (3), RTECS (6) and Sax (7). Information available (see appendices) available from the above-referenced databases includes various LD50s and TDLos. The chemical has been very well researched and its toxicity toward humans and other mammals is well established. The HSDB reports that the most serious toxic effect following ingestion is aspiration pneumonitis. Upon inhalation, the victim feels transient euphoria (alcohol-like intoxication), a burning sensation in the chest, headache, tinnitus, and nausea, followed by drowsiness, coma, and sometimes death.Inhalation may also result in irregular heartbeat, cardiac arrest, and central nervous system toxicity (3). Contact may burn the skin and eyes. The estimated fatal oral dose is 3 to 4 ounces in adult humans. Similar effects are found in small mammals (3). No information concerning toxicity toward aquatic species, birds, or plants could be found. Kerosene is a possible carcinogen (6).

Sax summarizes the data as: Poison by intravenous and intratracheal routes. Moderately toxic to humans by an unspecified route. Moderately toxic to animals by ingestion. Human systemic effects by ingestion and intravenous routes: somnolence, hallucinations and distorted perceptions, coughing, nausea or vomiting, and fever. Aspiration of vomitus can cause serious pneumonitis, particularly in young children. Combustible when exposed to heat or flame; can react with oxidizing materials. Moderately explosive in the form of vapor when exposed to heat or flame. When heated to decomposition it emits acrid smoke and fumes.

No reference standards were found in the literature.

Mineral Oil

Mineral Oil (syn. ADEPSINE OIL; ALBOLINE; BAYOL; IRGAWAX 361; PARAFFIN OIL; etc.), has the CAS number of 8012-95-1. It is manufactured by refining cruder lubricating oils to remove unsaturated or volatile compounds, as well as resins and compounds of nitrogen and sulfur (3). It is used as a food-safe lubricant on pans, rollers, belts and machinery in close proximity to many foods; as a defoaming agent in foods; as an ingredient in baby oils; as a veterinary pharmaceutical; in cosmetics; in animal feed; and in the flotation processing of mineral ores (3). It is a colorless, oily liquid. It is insoluble in water and alcohol, but is soluble in benzene, chloroform, ether, and carbon disulfide.

Information pertaining to kerosene has been obtained from the HSDB (3) and RTECS (6). Information available (see appendices) from the above-referenced databases includes various LD50s and TDLos. The chemical has been very well researched and its toxicity toward humans and other mammals is well established. The HSDB reports mineral oil is practically non-toxic, with a probable oral lethal dose of more than one quart for a 150 lb. person. Ingestion may cause nausea, vomiting and diarrhea. Aspiration of mineral oil may result in pneumonitis (3). No information concerning toxicity toward aquatic species, birds, or plants could be found. Mineral oil is potentially carcinogenic (6).

Sax summarizes the data as: A human carcinogen by inhalation which produces gastrointestinal tumors. A human teratogen by inhalation which causes testicular tumors in the fetus. Inhalation of vapor or particulates can cause aspiration pneumonia. An eye irritant. A laxative. Combustible when exposed to heat or flame. When heated to decomposition it emits acrid smoke and fumes.

No reference standards were found in the literature.

Pine tar

Pine tar (no synonyms) has the CAS number of 8011-48-1. Limited information concerning pine tar was found in the RTECS (6) database only. RTECS, however, reported only data from the National Occupational Hazard Survey, which reports only the number of employees known to be working with the chemical of interest. It also describes pine tar as a flammable liquid.

No reference standards were found in the literature.

Rosin acids, Tallow acids, Coco amine

Rosin acids, tallow acids, and coco amine, listed in the McNulty report (8), could not be found in the literature.

Although their toxicities could not be researched directly, it is likely that they are similar to other fatty acid compounds such as tall oil and oleic acid. The toxicities of both tall oil and oleic acid are discussed in this report.

Tall Oil

Tall Oil (syn. ACTINOL C; LIQUID ROSIN; TALL OIL ROSIN; UVDB; etc.) has the CAS number of 8002-26-4. It has a number of uses, including in the manufacturing of soap pastes; in greases, paints, and alkyd resins; linoleum; fungicides; rubber formulations; as the starting material for the manufacture of linoleic, oleic, and other fatty acid acids; and as a depressant in mineral ore flotation (3). It is manufactured by the digestion of pine wood pulp with sodium hydroxide and sodium sulfide followed by acidulation of the alkaline solution with sulfuric acid (3). It is a dark brown liquid with an acrid odor similar to that of burnt rosin (3).

Information pertaining to tall oil has been obtained from the HSDB (3) and Sax (7). Reported toxic effects range from dizziness, eye and skin irritation and burns, to possible suffocation (3). It may be poisonous if inhaled or absorbed through the skin (3). No other toxicological data were available from the sources searched.

Sax summarizes the data as: A mild allergen. A substance which migrates to food from packaging materials. Combustible when exposed to heat or flame; can react with oxidizing materials. When heated to decomposition it emits acrid smoke and irritating fumes (7).

No reference standards were found in the literature.

Wood Creosote

Wood creosote (syn. BEECHWOOD CREOSOTE; etc.), has the CAS number of 8021-39-4. It has a variety of uses, including as a antipyretic, styptic, or astringent; as a lubricant for die molds; as a waterproofing agent; as a wood preservative; as a veterinary parasiticide and disinfectant; as a human expectorant; and possibly in mineral ore processing (3). It is obtained by distillation of wood tar. It is not produced in the United States (3). The phenolic compound guaiacol, along with cresol and methylcresol are the primary compounds in wood creosote. It is an almost colorless or yellowish oily liquid with an aromatic, smoky odor and a burning caustic, phenolic taste (3). It is slightly soluble in water, miscible in alcohol and ether, and soluble in glycerol and glacial acetic acid (3).

Information pertaining to wood creosote has been obtained from the HSDB (3) and RTECS (6). Information available (see appendices) available from the above-referenced databases includes TDLo studies for rats only. Additional data can be found for coal creosote in the Appendices. Coal creosote is a Group 2A carcinogen (probable human carcinogen). Wood creosote is a probable carcinogen, although it has not been classified. As with coal creosote, ingestion of greater than 5% solution may result in oral burns. Eye exposure may result in severe burns. Contact with the skin or mucous membranes may cause intense burning and itching and gangrene in isolated instances. Deaths from large doses of creosote appear to be largely due to cardiovascular collapse. Death has occurred following ingestion of 7 grams by adults and 1-2 grams by children. Acute toxicity is believed to be caused by the presence of phenol or phenolic compounds (3). Laboratory bioassays indicate a generally moderate toxicity to aquatic organisms.

No Sax data summary was available for wood creosote.

No reference standards were found in the literature.

GLOSSARY AND LIST OF ACRONYMS

ACGIH — American Conference of Governmental Industrial Hygienists

ATSDR — Agency for Toxic Substances and Disease Registry

CAS — Chemical Abstracts Service

Chronic — Lifetime

DOT — U.S. Department of Transportation

e(0.7852[1n(hardness)]-3.490ug/L (4-day average concentrations - not to exceed, in ambient water quality)
For example, at hardnesses of 50, 100, and 200 mg/L as CaCO3, the 4-day average concentrations of this particular chemical are 0.66, 1.1, and 2.0 ug/L, respectively.

EPA — U.S. Environmental Protection Agency

EPA Human Carcinogenicity Group
Provisional cancer classifications - unit risk for carcinogenicity (based on life-time inhalation and oral exposure)

- Group A — Human carcinogen
- Group B1 — Probable human carcinogen - limited human evidence
- Group B2 — Probable human carcinogen - animal evidence/no or limited human evidence
- Group C — Possible human carcinogen - limited animal; inadequate/lack humans
- Group D — Not classifiable
- Group E — Evidence of noncarcinogenicity

HEAST — Health Effects Assessment Summary Tables

HHS — U.S. Department of Health and Human Services

HSDB — Hazardous Substances Data Base

IRIS — EPA's Integrated Risk Information System

LCLo — Lethal Concentration Low - the lowest concentration of a substance in air, other than LC50, which has been reported to have caused death in humans and animals. The reported concentrations may be entered for periods of exposure which are less than 24 hours (acute) or greater than 24 hours (subacute and chronic).

LC50 — Lethal Concentration Fifty - a calculated concentration of a substance in air, exposure to which for a specified length of time is expected to cause the death of 50% of an entire defined experimental animal population. It is determined from the exposure to the substance of a significant number from that population.

LDLo — Lethal Dose Low - the lowest dose (lower than LD50) of a substance introduced by any route other than inhalation, over any given period of time in one or more divided portions and reported to have caused death in humans or animals.

LD50	Lethal Dose Fifty - a calculated dose of a substance which is expected to cause the death of 50% of an experimental animal population. It is determined from the exposure to the substance by any route other than inhalation of a significant number from that population. Other lethal dose percentages, such as LD1, LD10, LD30 and LD99 may be published in the scientific literature for the specific purposes of the author. Such data would be published in the Registry if these figures, in the absence of a calculated lethal dose (LD50), were the lowest found in the literature.
LOAEL	Lowest observed adverse effect level
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
MF	Modifying factor
mg/L	milligrams per liter
MSDS	Material Safety Data Sheet
NAAQS	National Ambient Air Quality Standard
NIOSH	National Institute of Occupational Safety and Health
NOAEL	No observed adverse effect level
OSHA	Occupational Health and Safety Administration
PELs	Permissible Exposure Limits
ppb	parts per billion
ppm	parts per million
RfC	Inhalation provisional reference concentration
RfD	Oral provisional reference dose
RTECS	Registry of Toxic Effects Chemical Substances
SMCL	Secondary Maximum Contaminant Level
Subchronic	Portion of lifetime
TCLo	Toxic Concentration Low - the lowest concentration of a substance in air to which humans or animals have been exposed for any given period of time that has produced any toxic effect in humans or has produced a tumorigenic or reproductive effect in animals or humans.
TDLo	Toxic Dose Low - the lowest dose of a substance introduced by any route other than inhalation, over any given period of time to which humans or animals have been exposed and reported to produce any non-significant toxic effect in humans or to produce non-significant tumorigenic or reproductive effects in animals or humans.
TLVs	Threshold Limit Values

BIBLIOGRAPHY

ATSDR Publications:

1a U.S. Department of Health & Human Services, Toxicological Profile for Arsenic, February 18, 1992, Public Health Department, Agency for Toxic Substances and Disease Registry.

1b U.S. Department of Health & Human Services, Toxicological Profile for Cadmium, February 18, 1992, Public Health Department, Agency for Toxic Substances and Disease Registry.

1c U.S. Department of Health & Human Services, Toxicological Profile for Cobalt, 1991, Public Health Department, Agency for Toxic Substances and Disease Registry.

1d U.S. Department of Health & Human Services, Toxicological Profile for Copper, 1990, Public Health Department, Agency for Toxic Substances and Disease Registry.

1e U.S. Department of Health & Human Services, Toxicological Profile for Creosote, 1990, Public Health Department, Agency for Toxic Substances and Disease Registry.

1f U.S. Department of Health & Human Services, Toxicological Profile for Cyanide, February 18, 1992, Public Health Service, Agency for Toxic Substances and Disease Registry.

1g U.S. Department of Health & Human Services, Technical Report for Ethylene Glycol/Propylene Glycol, November 29, 1993, Public Health Service, Agency for Toxic Substances and Disease Registry.

1h U.S. Department of Health & Human Services, Toxicological Profile for Fluorides, Hydrogen Fluoride, and Fluorine (F), December 16, 1991, Public Health Service, Agency for Toxic Substances and Disease Registry.

1i U.S. Department of Health & Human Services, Toxicological Profile for Fuel Oils, November 29, 1993, Public Health Service, Agency for Toxic Substances and Disease Registry.

1j U.S. Department of Health & Human Services, Toxicological Profile for Lead, February 18, 1992, Public Health Service, Agency for Toxic Substances and Disease Registry.

1k U.S. Department of Health & Human Services, Toxicological Profile for Mercury, February 19, 1993, Public Health Service, Agency for Toxic Substances and Disease Registry.

1l U.S. Department of Health & Human Services, Toxicological Profile for Nickel, February 18, 1992, Public Health Service, Agency for Toxic Substances and Disease Registry.

1m U.S. Department of Health & Human Services, Toxicological Profile for Polycyclic Aromatic Hydrocarbons, 1990, Public Health Service, Agency for Toxic Substances and Disease Registry.

1n U.S. Department of Health & Human Services, Toxicological Profile for Silver, 1990, Public Health Service, Agency for Toxic Substances and Disease Registry.

1p U.S. Department of Health & Human Services, Toxicological Profile for Zinc, February 19, 1993, Public Health Service, Agency for Toxic Substances and Disease Registry.

2 HEAST Reference:

U.S. Environmental Protection Agency, Health Effects Assessment Summary Tables - Annual Update, March 1993.

3 HSDB Reference:

U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Library of Medicine, Toxicology Data Network, MEDLARS online database system - Hazardous Substances Data Base, updated periodically.

4 IRIS Reference:

U.S. Environmental Protection Agency, Integrated Risk Information System, online computer system updated periodically.

5 MSDS Reference:

Dow Chemical Company, Midland Michigan, Material Safety Data Sheet, December 1993.

6 RTECS Reference:

U.S. Department of Health and Human Services, Public Health Service, National Institues of Health, Library of Medicine, Toxicology Data Network, MEDLARS online database system- Registry of Toxic Effects Chemical Substances, updated periodically.

7 Sax, Irving J., Dangerous Properties of Industrial Materials (7th Edition), 1989.

8 McNulty, T.P. and Associates, Inc., Constituents from Mining and Processing of Ores, May 1993.

9 EPA "Gold Book" Reference:

U.S. Environmental Protection Agency, Quality Criteria for Water - 1986, Updates 1 and 2 Included, May 1987, EPA 440/5-86-001.

OTHER SOURCES INVESTIGATED

Ankley, Gary, U.S. EPA, Duluth, Minnesota Laboratory, personal communication regarding sediment toxicity, 1993.

Herbert, Charles D., Ph.D, NTP Technical Report on Toxicity Studies of Cupric Sulfate, July 1993, United States Department of Health and Human Services, Public Health Service, National Institutes of Health.

Eisler, William, U.S. Fish and Wildlife Service, personal communication regarding contaminant hazard reviews, 1993.

Environmental Science and Engineering, Litigation Technical Support and Services, Rocky Mountain Arsenal, Biota Remedial Investigation, Draft Final Version, January 26, 1989.

Michigan State University Plant Toxicology Laboratory, East Lansing, Michigan, personal communication regarding plant toxicology, 1994.

Tillit, Don, U.S. Fish and Wildlife Service, Fisheries Contaminant Research Center, Columbia, Missouri, personal communication regarding fisheries contaminant research, 1993.

U.S. Environmental Protection Agency, Public Access Online Library System, 1993-94.

U.S. Environmental Protection Agency, Record of Decision, Anaconda Co. Smelter NPL Site, Flue Dust Operable Unit, Anaconda, Deer Lodge County, Montana, September 1991.

U.S. Environmental Protection Agency, Record of Decision, California Gulch, Leadville, Lake County, Colorado, Operable Unit 1 - Yak Tunnel, undated.

U.S. Environmental Protection Agency, Record of Decision, Clear Creek/Central City Superfund Site, Operable Unit #3, Gilpin and Clear Creek Counties, Colorado, September 30, 1991.

U.S. Environmental Protection Agency, Record of Decision, Eagle Mine Site, Operable Unit 1, Eagle County, Colorado, undated.

Wuerthele, William, U.S. EPA Region VIII, personal communication regarding ambient water quality criteria, 1993.